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A PRACTICAL JOURNAL BUILT ON MERIT

# EDITORIAL

## SURGICAL RESIDENCIES AND APPROVED SPECIALISTS

AFTER 1941–1942 no physician will be eligible to take the examinations held by some of the American Boards for certification in a specialty unless he has held for a prescribed time a residency approved by the American Medical Association. Many physicians, mainly the younger group, claim that there are not sufficient residencies in this country to meet the demand, and that many capable men are therefore denied the opportunity to equip themselves for specialization.

For a long time we, too, had the same impression. In fact, we went further in our speculation—and thought that possibly in ten or twenty years, if only certified men could be specialists, the country would be faced with a shortage of competently trained men; there might not be enough recognized specialists in surgery (in all its branches) to administer to the country's needs.

However, the August 26, 1939 number of the *Journal of the American Medical Association* lists the various residencies and fellowships in approved hospitals or services in the United States, and the list shows that there is no apparent shortage. Residencies in specialties, as defined by the Council, are straight services of one or more years, following an approved internship. We computed the approved residencies in surgery, gynecology, and obstetrics and added the combined residencies in obstetrics and gynecology. Our figures may be slightly in error, but not enough so to affect the conclusions.

In surgery, 303 physicians were residents in August, 1939; in gynecology, there were twenty-one; in obstetrics the total was seventy-four; and in the combined field of obstetrics and gynecology, the number was 127. Therefore, assuming that this number will not decrease (the probability is that it will increase from year to year as further facilities are made available), each year about 300 men will be eligible to take their American Board examinations in surgery, and about 125 in obstetrics and gynecology. When these numbers are multiplied by ten or twenty, we arrive at the total of certified men at the end of ten or twenty years, with some allowance for decreases due to death or retirement. It is apparent that there will be no dearth of specialists in this country. With the constant addition of approved residencies as the years pass, the opposite may be the fact.

Every young man in training as an intern cannot be successful in getting a residency—not, at any rate, at the present time. Every physician who has the desire or inclination cannot become a certified specialist. Which may be a good thing. Otherwise, imagine the state of the profession if the United States had 120,000 specialists and a mere handful of general practitioners!

A grave responsibility rests on the shoulders of the surgical directors who select residents for the available places. They must make sure that the men they choose have the training, aptitude, possible manual dexterity, and moral fiber for the job. The right man should not be denied because those in power make wrong or poor selections.

T. S. W.

# ORIGINAL ARTICLES

## PERIPHERAL NERVE INJURIES, WITH RESULTS OF EARLY AND DELAYED SUTURE

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### INTRODUCTION

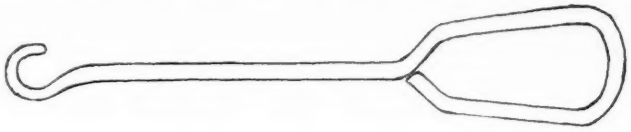
IN the present day, with the increase in highway and industrial accidents, the subject of treatment of peripheral nerve injuries is of great interest to all surgeons. During the World War I was very fortunate to be associated with the British Army and was assigned to Alder Hey Military Hospital, Liverpool, where I had the great pleasure of working with Mr. T. R. Armour, F.R.C.S. and Sir Robert Jones for some seventeen months. Ultimately I taught this work of handling nerve injuries in that hospital. Little of a practical nature has been written on the subject of the treatment of nerve injuries, and for that reason, based on my experience during the War and in civil practice, I feel it advisable to discuss this important matter.

### GENERAL CONSIDERATIONS

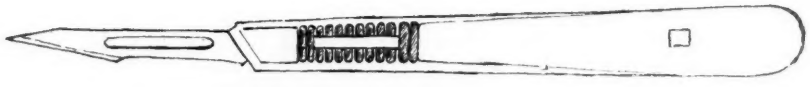
When we first went into the surgery of peripheral nerve injuries during the War we were of the opinion that a sensory nerve could be made to bridge a motor gap; in other words, that a gap in the ulnar, median or radial at or distal to the elbow could be bridged by using the musculocutaneous; likewise that the sciatic, posterior tibial, anterior tibial and peroneal could be bridged by using the long saphenous. After many attempts along this line we found that the interposed sensory nerve did not become converted into a functioning bridge in a motor nerve. Under those circumstances, we had to devise other means of overcoming our difficulties in nerve suture.

We found that end-to-end suture gave the only good results and that we must suture only the sheath (not invading the neurons with suture material); that nerve suture must be done *without tension* or it would pull apart with disastrous results; that a nerve gap could be closed by stretching the proximal or distal portion of the severed nerve (or both), to break up perineural adhesions, and by flexing the adjacent joints to shorten the nerve path; that generally speaking the sutured nerve must be kept in this protected and relaxed position by a cast or other fixation for a period of *forty-five days* after suture. We could then extend the flexed joint gradually over a period of two to three weeks or even longer, a few degrees at a time, until the joint could be brought to normal position, thus allowing a gradual stretching of the healed nerve. We learned that the sensory fibers (of mixed sensory and motor nerves, like the radial, median and ulnar) are the first to show unmistakable signs of regeneration following nerve suture. Then voluntary muscle control appears, and last appears the positive reaction of the reinnervated muscle to faradism (as tested by the Bristow coil); in other words, the process of mixed nerve regeneration is (1) return of sensation; (2) voluntary control; (3) reaction to faradism, in that order.

I wish to emphasize that to do nerve surgery, as well as to diagnose nerve lesions correctly, one must know the anatomy intimately, or else decline to undertake this delicate and important form of surgery. I am not going into the detailed anatomy in the following regional comments, but I assume that the reader will study this in his own time and at his discretion.



BUTTONHOOK FOR LIFTING NERVE



KNIFE FOR NERVE DISSECTION -

FIG. 1.

In the *diagnosis of peripheral nerve lesions, and there only*, faradism is of use; it cannot be used for treatment after nerve suture. Sinusoidal galvanism is of great use, however, in the treatment of the affected muscles after nerve suture, but is of *no use* in diagnosis of nerve lesions. In other words, if before operation you wish to discover whether a nerve is severed, partially or completely, you use faradic stimulation above the site of the nerve lesion, over the muscles supplied by the nerve, and depending upon the strength of the response to this electrical stimulus you are able to determine the extent of nerve injury, whether partial or complete. In addition, you may map out the area of sensory impairment in testing for touch, pain (pinprick), heat and cold, and test the voluntary muscle actions. You may also note any muscle atrophy and any trophic changes in the member, to complete your diagnosis. If you are not conversant with peripheral nerve injuries, the above mentioned tests and the detailed anatomy, you should call in a surgeon or neurologist familiar with this type of work.

## SURGICAL TECHNIQUE

In incising over the nerve lesion (new or old) I advise you to make your incision in line with the course of the nerve, and the incision should give a generous and sensible exposure of both the proximal and the distal ends. It is always good judgment to pick up the proximal nerve (normal, well above the wound or scar) and after having identified it, dissect distally to the point of nerve severance, likewise the distal end. This rule will save much trouble and should be applied to new and especially to old cases of nerve trauma, because by exposing and following the normal appearing nerve tract above and below the lesion, you are in a position to differentiate the abnormal or pathologic nerve tissue from the normal, as you dissect.

Do not keep at one end too long at a time, but change back and forth between

the proximal and the distal. This will avoid fatigue and give you a change of visual field and avoid confusion of structures. the nerve ends, then *suture the sheath only*, using the very finest of needles and a double or triple zero catgut. (Fig. 2.) Use

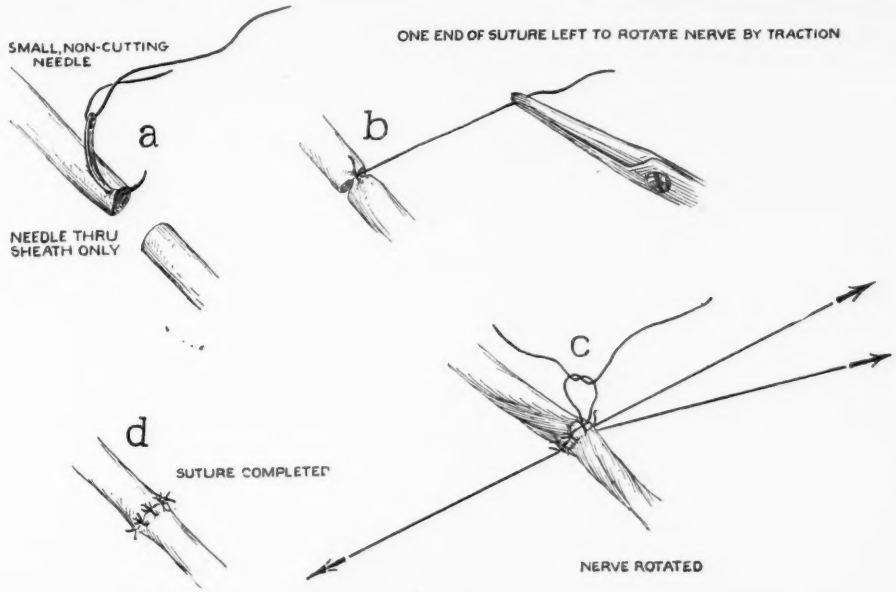



Fig. 2.

Peripheral nerve surgery is difficult and tedious at best. Do all your nerve work by *sharp dissection* only; blunt dissection, ripping, rubbing or tearing structures in this work, once the nerve has been brought into view, is a serious mistake. By passing a curved smooth probe or buttonhook under the nerve you have full control of it. interrupted sutures placed close together, being sure that all exposed neurons are replaced within the reconstructed sheath, because if one or more of the axis cylinders is left exposed, a "wild growth" of these cylinders develops later, resulting in a painful neuroma, or causalgia. This is particularly demonstrated in the median

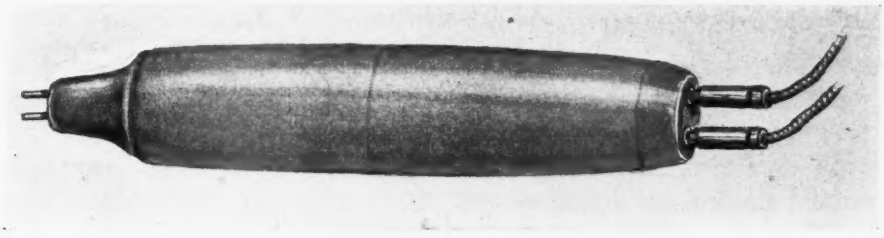


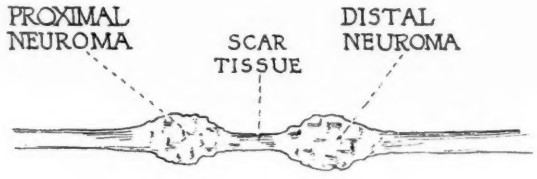
Fig. 3.

You should avoid bruising or picking up the nerve, even with blunt forceps, any more than necessary. Use a small, very sharp pointed scalpel in nerve dissection. (Fig. 1.)

In dissecting out a *freshly cut nerve*, find the severed ends, and if they are ragged, trim them off flat so that the sheath and neuraxons are clean cut and can be approximated end to end. Having prepared nerve in the arm, and in the internal popliteal in the leg; I saw one case in the ulnar (causalgia) during my War service.

There are two schools of thought in the use of material for nerve suture; one advocates the use of very fine silk, the other favors fine catgut. Personally, I always use the catgut, for silk never absorbs but produces some fibrosis of the tissues, a

neuromatous formation, and a poor or unsatisfactory result; in using very fine tanned catgut sutures *without tension*, the




NEUROMA

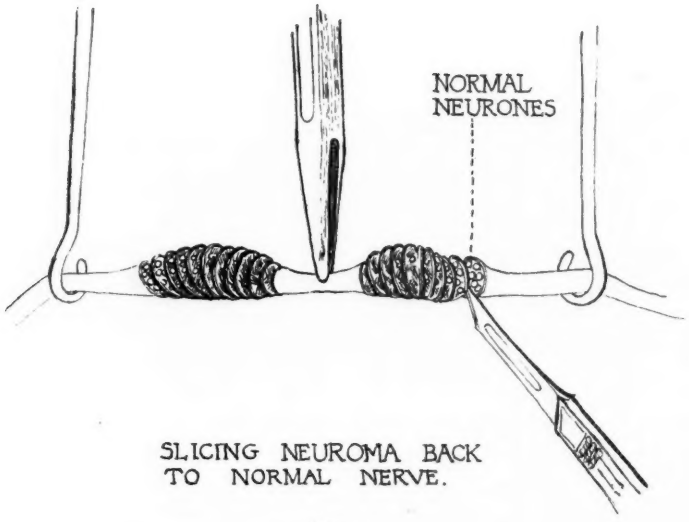



Fig. 4.

catgut is absorbed in forty-five days or sooner and produces no fibrosis. This was very definitely proved by the British surgeons during the War. Since that time in civil practice I have seen a number of cases where silk was used, with failures as results.

In *delayed repair of severed nerve*, a month or more after the original trauma, you will find on exposing the nerve, a gap in the nerve substance where severed. The gap is filled with scar tissue and there is a neuromatous bulb at each end of the cut nerve, with the bulb on the proximal end usually the larger. In testing the nerve function, apply a sterilized faradic point to the nerve (under low power) above the lesion, and if no muscle response occurs distal to the suspected lesion, you know the nerve is severed. (Fig. 3.) You are fully justified in cutting through the scar tissue in the nerve gap; then, picking up one end at a time, cut transversely across the nerve end, slicing at close intervals until you have exposed and seen the normal axis cylinders in cross-section lying like a closely packed bunch of cables in the nerve sheath. (Fig. 4.) In the operating room we use the faradic point with the tissues exposed. Therefore we cannot use ethylene anesthesia safely, and accordingly, we rely on ether anesthesia.

Of course, you must be careful in this nerve trimming process that you are not placed in the embarrassing position of making such a large "nerve gap" that you are unable to approximate the ends of the

nerve for suturing. To close the gap, as previously described, you have recourse to stretching the nerve as well as flexing making the new bed as smooth as possible, because with a careful closure of the nerve sheath there will be no troublesome ad-

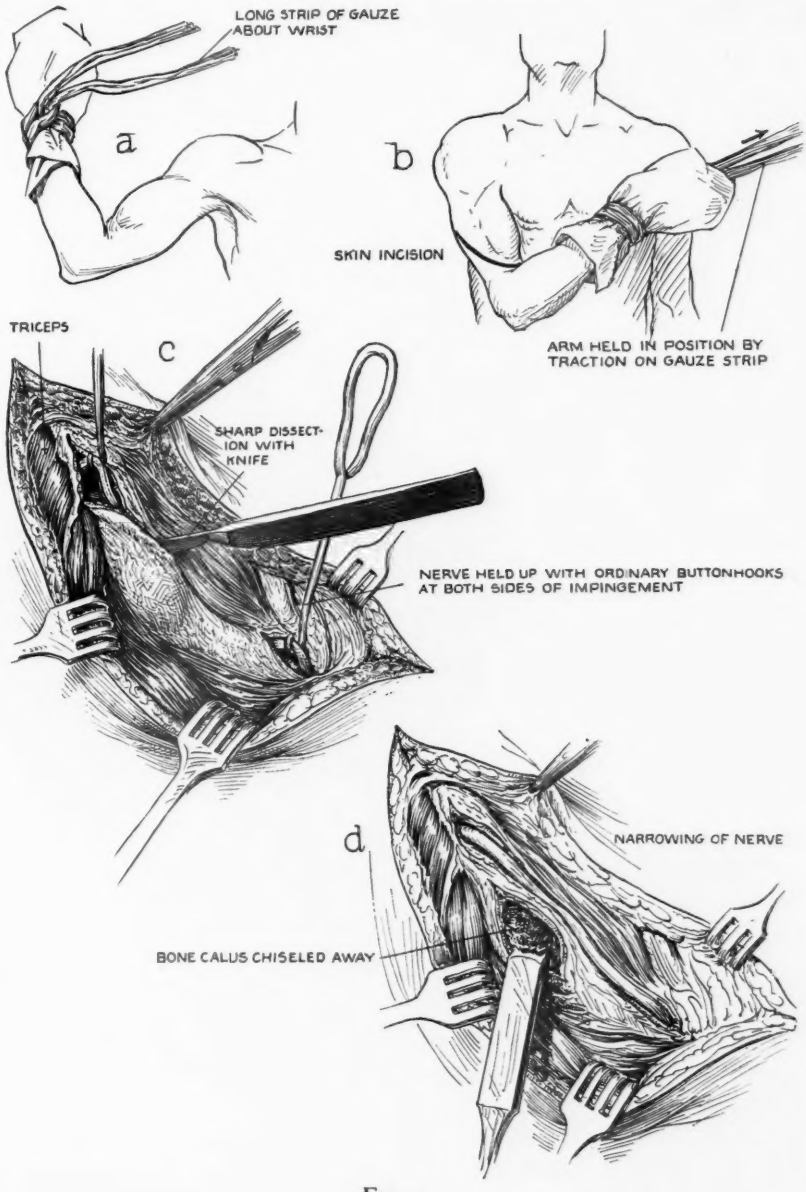



Fig. 5.

the adjacent joints as you trim the nerve, in order to be sure you can suture the nerve ends together *without tension*. In delayed nerve suture there is no necessity for protecting the reconstructed nerve by placing the fat or fascia around the nerve suture; you need simply debride the tissue bed where the repaired nerve will be placed, removing all tags of tissue and hesions. *Never* transplant a nerve *through* a muscle belly, as the muscle fibers will attach themselves to the nerve sheath and cause a constriction with a resultant causalgia or dysfunction which will require another operation and placing the nerve in a smooth bed to avoid adhesions.

Even a delayed nerve suture can lead to a good functional recovery if the work is

properly carried out, as shown in the later case reports.

Nerve suture should be done under general anesthesia and it is often advisable to use a tourniquet to render the dissection and repair easier in a bloodless field. In the arm (above the elbow) you can safely use a tourniquet up to one hour, and above the knee for two and one-half to three hours. On the arm we prefer to use a Martin

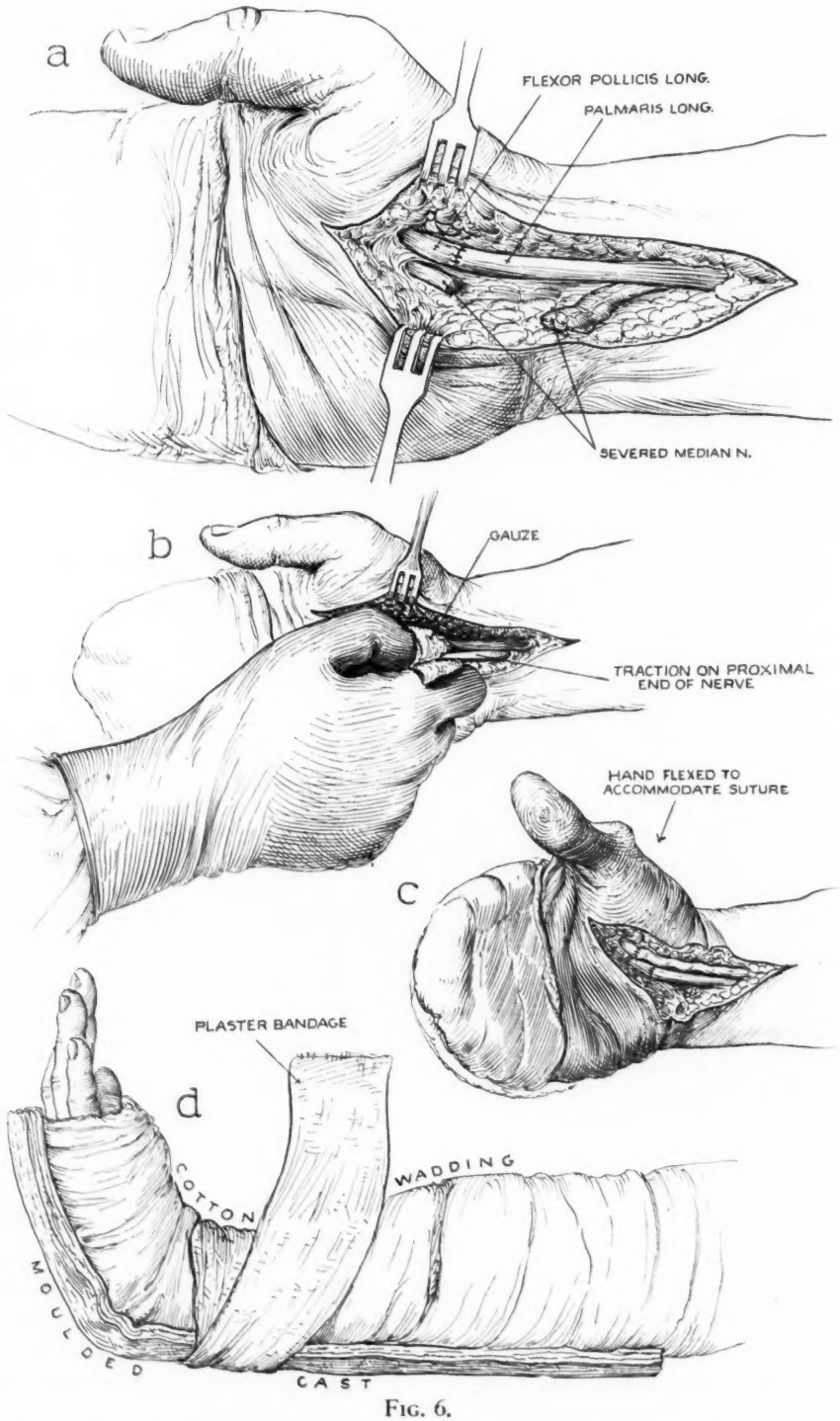



Fig. 6.

rubber bandage, for it cuts in on the soft tissue less than an Esmarch does; on the thigh an Esmarch is best.

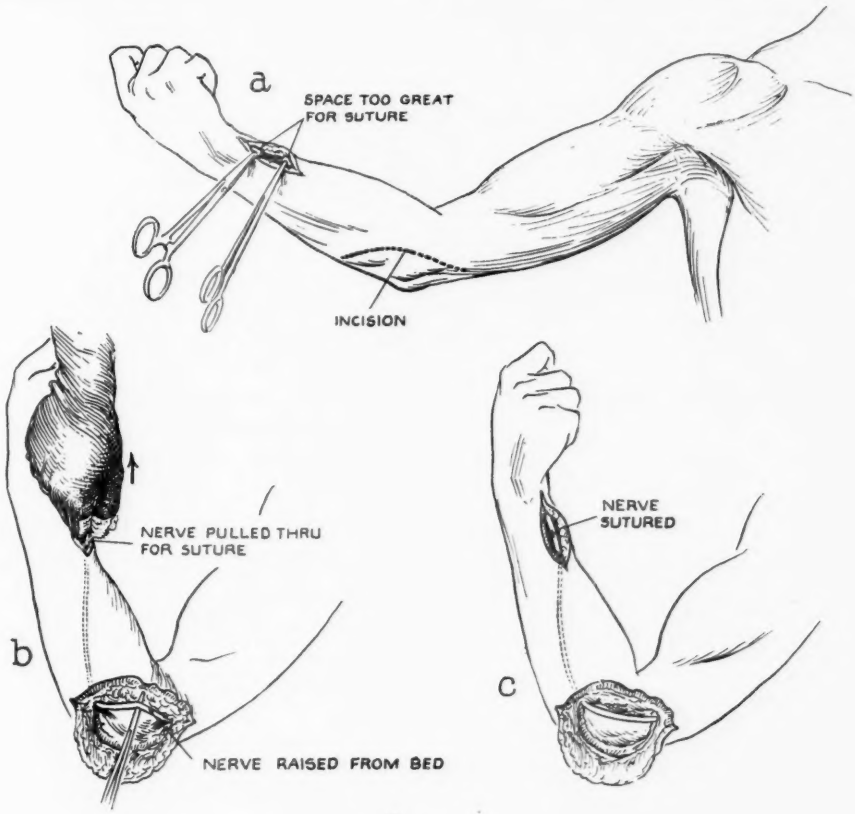



Fig. 7.

### COMMENTS ON INDIVIDUAL NERVES

1. *Brachial Plexus.* Where the roots are avulsed or severed at their emergence from the spinal foramina, suture is usually impossible, or useless if attempted. Where severed in the base of the neck, suture can be done with more or less success. In the War we learned to resect a portion of the clavicle and ligate the subclavian vein in order to gain sufficient exposure to allow a successful suture of the brachial plexus. Injuries to the plexus are seldom encountered in civil practice.

2. *Circumflex Nerve.* Suture of this nerve is very difficult and as a rule not successful, due to its deep position under the shoulder muscles. If suture is attempted, the approach must be from behind the shoulder (refer to anatomy).

3. *Musculocutaneous Nerve.* In my War and civil practice I never saw an injury to the musculocutaneous nerve (except in amputations or hopelessly destructive lesions of the arm). This nerve makes its exit out of the belly of the biceps at the junction of the lower and middle thirds of this muscle, supplying the biceps brachii, coracobrachialis anticus. Distal to that it is purely a sensory nerve, hence the rarity of any important injury to it.

4. *Musculospiral Nerve.* The usual location of a traumatic lesion of this nerve is in the arm at about the junction of the middle and lower thirds of the humerus where it is often injured in fractures at this point. The nerve may be severed, caught between bone fragments, or later incorporated in the callus with a resultant paralysis. (We have operated in two unusual cases, one a simple nail puncture, another a sliver of steel.) The surgical approach to this nerve is started at a point

10 cm. proximal to the external condyle and the incision follows a descending line from the back of the intermuscular septum

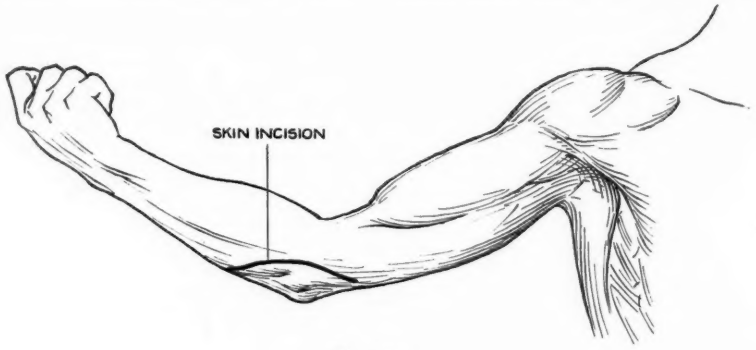



Fig. 8a.

distally and anteriorly to the antecubital space.

Like all nerve sutures, suture of the musculospiral must be done without tension. Hence the elbow should be maintained at a right angle for forty-five days after operation, preferably in a cast, with a window cut for redressing of the incision;

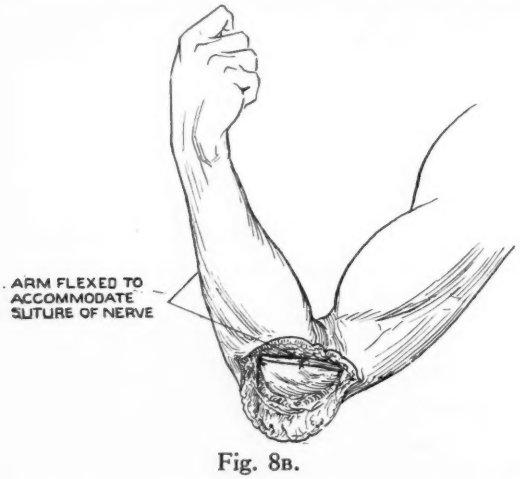



Fig. 8b.

during the period of recovery, the wrist drop should be corrected by a cock-up splint until voluntary muscle action has recovered sufficiently to accomplish hyperextension of the wrist and fingers. After the first forty-five days the elbow should be extended gradually for a period of two or three weeks or longer, until it can be fully straightened, thus avoiding any abrupt stretching of the healing nerve. On cast removal, use sinusoidal galvanism treatments three times a week for six to eight months if necessary, over the affected muscles, to maintain muscle tone and volume until such time as the nerve regenerates and functions; even after that you should continue sinusoidal treatment until you are satisfied that you have gained all you can in recovery.

In immediate suture of the musculospiral the results are good, with sensory function appearing in the web between the thumb and index finger dorsally in about two or three months after nerve suture, and voluntary muscle response (extension of wrist and digits) in about four to six months. Faradic response usually shows in about six to eight months and improvement continues for a year, occasionally longer, after suture. Length of temporary total disability depends on the patient's occupation, of course. The functional cure

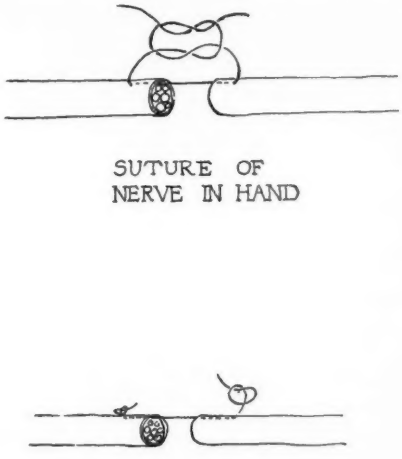



Fig. 9.

is complete when the patient is able to place the dorsal surface of his forearm and hand on a table and extend all the fingers until he touches the tips to the table.

elbow completely, suturing the nerve end to end in that position. We found we could close a gap of 2 inches (maximum) by wrist flexion, combined with elbow flexion.

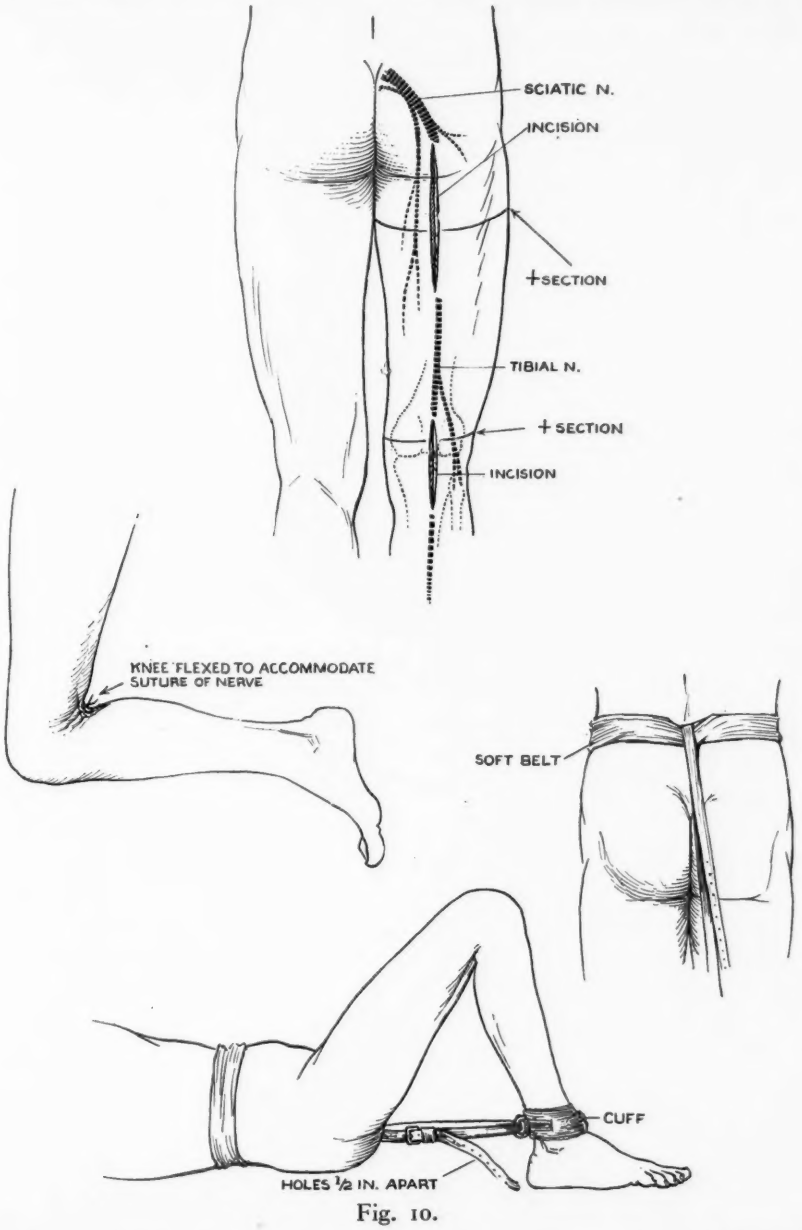



Fig. 10.

5. *Median Nerve.* This is most frequently injured at and just proximal to the wrist level, as seen in civil practice, although I have seen it injured at or near the elbow in the War, due to gunshot wounds.

In the War injuries we found we could bridge a gap in the median nerve at the wrist or elbow by stretching the proximal end of this nerve and flexing the wrist or

This position is maintained during nerve suture and after closure of the tissues in a cast for forty-five days. Then the wrist and elbow are extended about an inch at a time until the member is brought back to normal. After cast removal, sinusoidal galvanism is given about three times weekly, as described under the musculospiral nerve technique.

It is surprising how small a wound at the wrist can involve the median nerve; anatomically, it lies alongside of and slightly deeper than the palmaris longus tendon, so the nerve can be injured easily in wrist wounds. Whenever you are faced with a volar wound at the wrist, a careful test *and exploration* should be made for both median and ulnar nerve injuries. These nerves are very important in the function of the hand, and the sooner a nerve repair is made the better the end results will be. If six hours or more have elapsed since injury occurred, you had better delay nerve suture for fear of infection, unless you feel you can thoroughly debride the tissues. Otherwise, wait a period of about two to three weeks in dealing with a cleanly healing wound; or two to three months after infection subsides, if infection does occur. A wound infection is disastrous, of course, in its effect on a nerve suture, when repair is attempted under such circumstances.

A return of nerve sensation usually can be detected in an average time of two to three months after the immediate suture, and progresses to complete or almost complete sensibility. The return of the motor function is slower, usually being evident in five to six months and progressively improving. Voluntary muscle control, then Faradic response appears. Sinusoidal galvanism, massage and exercises should be continued for six to eight months at least, and usually longer if the patient will permit. The maximum of recovery usually takes place in one to one and one-half years. Disability depends on the patient's occupation.

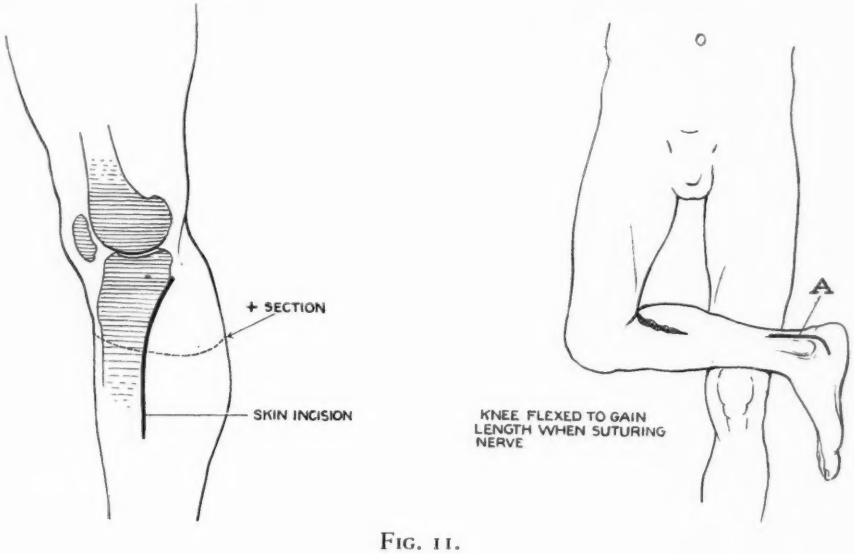



Fig. 11.

Median nerve causalgia is an occasional result of injury to this nerve when untreated or improperly sutured, and is very painful and disabling.

6. *Ulnar Nerve.* This is severed or injured most commonly at the wrist level, and next most commonly at the elbow. At the wrist it lies superficially enough, close to the ulnar vessels and flexor carpi ulnaris tendon so that it is injured just as easily as the median nerve. In the repairing of the flexor carpi ulnaris or the stopping of hemorrhage from the ulnar vessels, the ulnar nerve severance is frequently overlooked by a careless exploration, and a late repair is necessitated. The ulnar nerve paralysis is very crippling to the hand, and fingers, not only in the numbness resulting but in the muscle effects, hence the importance of immediate repair which yields so much better a result than delayed nerve suture.

The technique (Figs. 7 and 8) is the same as in median nerve suture at the wrist,

except that if a large nerve gap has to be closed, one must gain length of nerve by flexing the wrist completely and, if necessary, making a second incision at the medial aspect of the elbow and transplanting the nerve forward of the condyle, then flexing the elbow; a considerable gap (at least 2 inches) in the nerve at the wrist can be closed by traction on the proximal nerve end, and by right angle flexion of the wrist and elbow. The after treatment is the same as described for the musculospiral and median nerves (forty-five days in cast, followed by massage and sinusoidal galvanism three times a week, etc.). Sensation begins to return in two to four months in successful suture, and motor voluntary functions begin to show in four to six months as a rule. Recovery is slow in the ulnar, taking one and one-half to two years, during which time medical treatment (such as sinusoidal galvanism and massage) and observation preferably should be continued.

The more proximal ulnar lesion, at the elbow, recovers more slowly after nerve suture, as the neuraxons have so much further to regenerate. A fairly good return of sensory function can be expected, but return of motor function is more uncertain and incomplete. The earlier the nerve suture can be done, the more complete is the eventual recovery, as muscle atrophy and fibrosis and contracture of the ring and little fingers do not have a chance to become apparent.

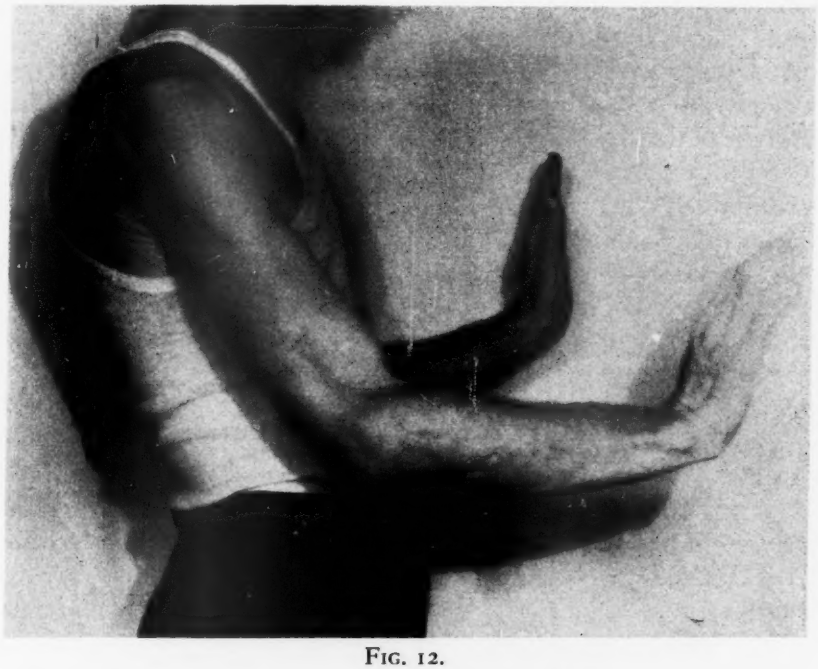


Fig. 12.

7. *Nerves of the Hand and Fingers.* In the hand they can be sutured with success if carefully done, though this is very difficult work. On the branches of the median and ulnar nerves, a triple zero Pyoktannin catgut strand can be run through the sheath of the proximal nerve end and the distal nerve end, drawing the ends together and holding in approximation by a knot close to the sheath at each end of the suture. We have done this successfully on the median nerve branch to the middle finger in one case, and to the thumb in another case, and these patients recovered skin sensation over that nerve distribution.

The nerves of the fingers (the digital branches of the median and ulnar nerves) are so small that suturing them is out of the question. They are large enough so that a painful neuroma in the scar can be found and dissected out quite readily, using a rubber band tourniquet to render the field

of dissection bloodless. One can resect completely such a finger neuroma without attempt at nerve suture, and still leave good function in the finger.

8. *Sciatic Nerve.* This is one of the most commonly injured structures in the lower extremity, between the hip and the thigh. Injury to the sciatic nerve is seen only occasionally in civil practice, although it was seen quite commonly during the World War in my experience. We found that any attempt to suture a sciatic lesion proximal to the gluteal fold was impractical, because anatomically this nerve begins to bifurcate at the lower border of the quadratus femoris muscle and interlacing this area is a vast arterial supply which surrounds the nerve at this junction. Attempting nerve suture here would result in extensive hemorrhage and possible death. The reader can study out the detailed anatomy of this region if he desires.

Between the gluteal fold and the popliteal space this nerve can be sutured, and can be brought together where there is a gap of as much as 5 inches. This is accomplished first by making a medial incision, exposing both nerve ends, using strong traction on the distal portion of the nerve and thus gaining a stretch of 2 inches (and there is no harm done in stretching gently the proximal end); then the knee can be flexed to a right angle or more, thus giving the operator another 3 inches of nerve. The ends can be approximated and sutured without tension in this position, the limb maintained thus for at least forty-five days by the use of a belt around the abdomen, another round the ankle, and running between the belts a leather strap and buckle with belt holes one-quarter inch apart. At the end of forty-five days, you can begin to let the knee straighten and the leg come down, one hole at a time, and you will find that the natural weight of the lower extremity will stretch the nerve gradually without tearing the sheath sutures apart. Meanwhile sinusoidal galvanism can be used on the muscles below the seat of the lesion, the treatment being rendered at least three times a week. This will maintain the muscle tonicity and volume until such time as the nerve has regenerated sufficiently to resume its responsibilities. I wish to emphasize again that sinusoidal galvanism is used (in these treatments after nerve suturing) and not Faradism (the latter is useful only in diagnosis).

Prognosis: A successful suture requires one and one-half to two years for recovery. Disability runs nine to twelve months. There is always a temporary residual foot drop in these cases, which can be controlled by the use of a prosthetic shoe holding the foot at right angles while walking.

9. *Internal Popliteal Nerve.* Keep in mind that some 4 inches above the popliteal space the sciatic makes its division into the external and the internal popliteal nerves, the internal passing down through the popliteal space under the inner head of the gastrocnemius to perforate the soleus muscle and lie to the inner side of the leg, passing down to the ankle where it bifurcates again into the two plantar branches.

My reason for going into this detail is that if a lesion of this nerve exists below the popliteal space you must not try to reach the nerve (at operation) by placing the patient on his abdomen and going between the two bellies of the gastrocnemius; but have the patient placed on his back, cross the affected limb over the other (Fig. 11) and make incision anterior to the gastrocnemius, drawing the belly of the inner head to one side, thus exposing the soleus muscle; pass through the fibers of the soleus and you can reach the internal popliteal nerve easily. A gap of at least 2 inches in this nerve can be closed by flexing the knee, the same as one would do in a high sciatic.

Prognosis: One to two years for full recovery. Disability endures for five to six months, and then a prosthetic shoe holding the foot at right angles should be worn until full recovery.

10. *External Popliteal and Peroneal Nerves.* Keep in mind that the anterior

tibial nerve passes around the outer side of the head of the fibula, and as it does so, it breaks up into branches to supply the extensor muscle group, the peroneal division supplying the peroneus longus and brevis. It separates from the external popliteal in the popliteal space. It is therefore always advisable to explore these branches where there is a wound in the popliteal space.

Prognosis: One to two years for recovery. Disability five to six months with a prosthetic drop foot shoe.

11. *Anterior Crural Nerve.* This nerve I have seldom found to be injured in my experience in civil practice, and I saw only two cases of this lesion during my War experience; in fact, the only region in which this nerve can be affected is the groin or just distal to it. A wound in this location is almost certain to involve also the closely associated femoral artery and vein, and usually death results from hemorrhage or gangrene of the leg. In the two War cases above mentioned, curiously enough, through some unusual direction of the striking force, the anterior crural nerve alone was involved and both patients lived. In civil practice, we have seen one case where an ice pick penetrated the femoral region and severed the nerve and vessels, resulting in gangrene and amputation. Prognosis is one to two years for recovery. Disability one to two years.

Fig. 13. Case 1.

## CASE REPORTS

Following are brief descriptions of some actual cases in which we have performed early or late nerve suture, illustrating the technique, after-care, progress and results in the more common peripheral nerve lesions:

Case 1. Suture of the musculospiral nerve above elbow (two months after injury). End result at thirteen months.

C. I., age 35, a married truck washer, was injured June 8, 1937. A truck tire rim shattered and caused a small puncture wound above the right elbow; the wound was repaired elsewhere and was seen by us eight hours after injury. Complete musculospiral paralysis was present.

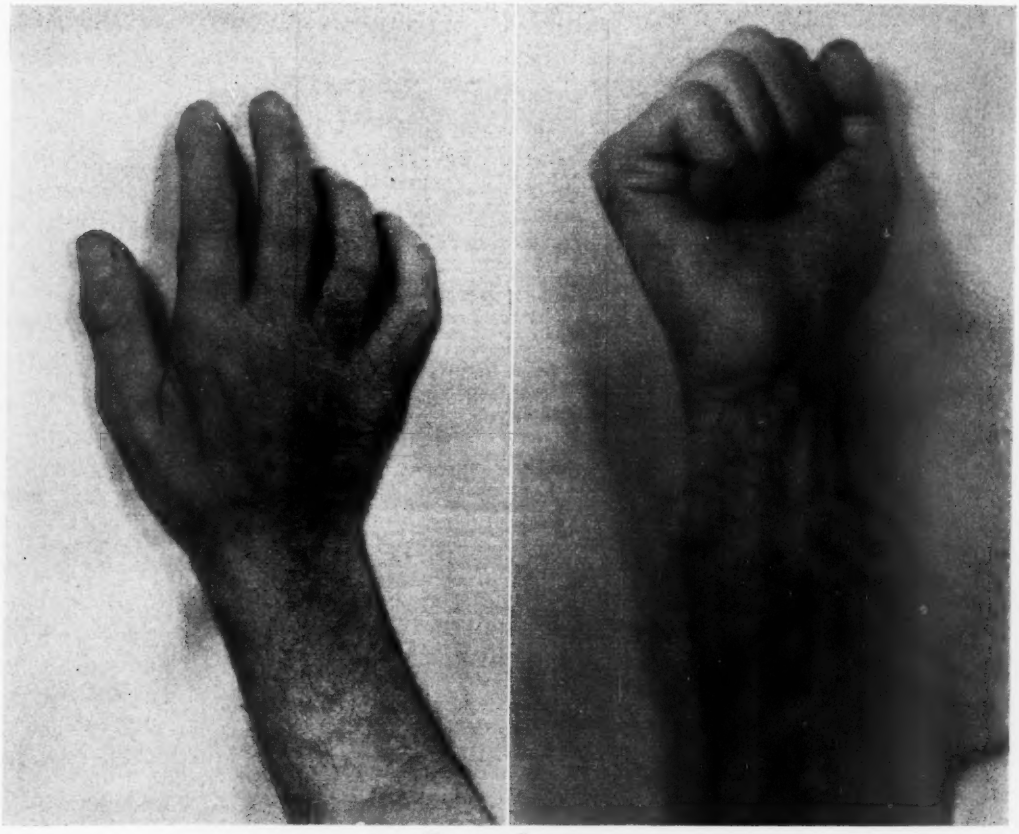


Fig. 14. Case vi.

We used a cock-up splint for the wrist, waited until August 14, then we operated, found the nerve severed 2 inches above the elbow, scarred and bulbous, with an encysted steel fragment near it. The nerve was sutured, the arm kept in a cast with elbow flexed until forty-five days postoperative, then the elbow was gradually extended. Sinusoidal galvanism, heat lamp and massage once weekly were started September 28, 1937. By October 22, 1937, there was weak response in the wrist and finger extension to sinusoidal stimulation, but no voluntary muscle action. By November 29, a flicker of voluntary extension appeared at the wrist. Hyperesthesia was present over the thumb web dorsally by January 26, 1938. By March 22, 1938 he could extend the wrist 50 degrees. Sinusoidal galvanism and massage were continued until March 31, 1938 (eight months postoperative).

Disability was prolonged due to a delayed union in a compound fracture of the right leg caused in the same accident, and the fact that the patient found difficulty in obtaining a job. He actually returned to work July 1, 1938, about thirteen months after injury, as a W. P. A. laborer, also working on his free days as a washing, greasing and gasoline attendant in a garage, and continued this work. Our last examination, September 7, 1938, showed full motion of elbow, wrist (70 degrees extension) and fingers, slight impairment in strength of thumb extension (though full range), slightly impaired and hyperesthetic sensation dorsally at thumb and thenar web. He thinks he is still improving in motion.

Case ii. Delayed lysis (seventeen days) of pinched musculospiral nerve.

L. R., age 18, single, schoolgirl, was injured May 10, 1936 and was first seen by us eleven days after injury. She had sustained a transverse fracture of the left humerus at the junction of the middle and lower thirds, with complete musculospiral nerve paralysis, total wrist drop, and fracture displaced. At operation on May 26, 1936, seventeen days after

injury, we found the nerve caught and constricted between bone fragments; it was freed, the fracture reduced with an intramedullary beef bone peg, and the nerve transplanted to extramuscular position. A cast with elbow flexed was applied. Two and one-half months postoperatively, we started sinusoidal galvanism twice weekly, with heat and massage. Three and one-quarter months postoperative we removed the posterior mould when bone union was firm.

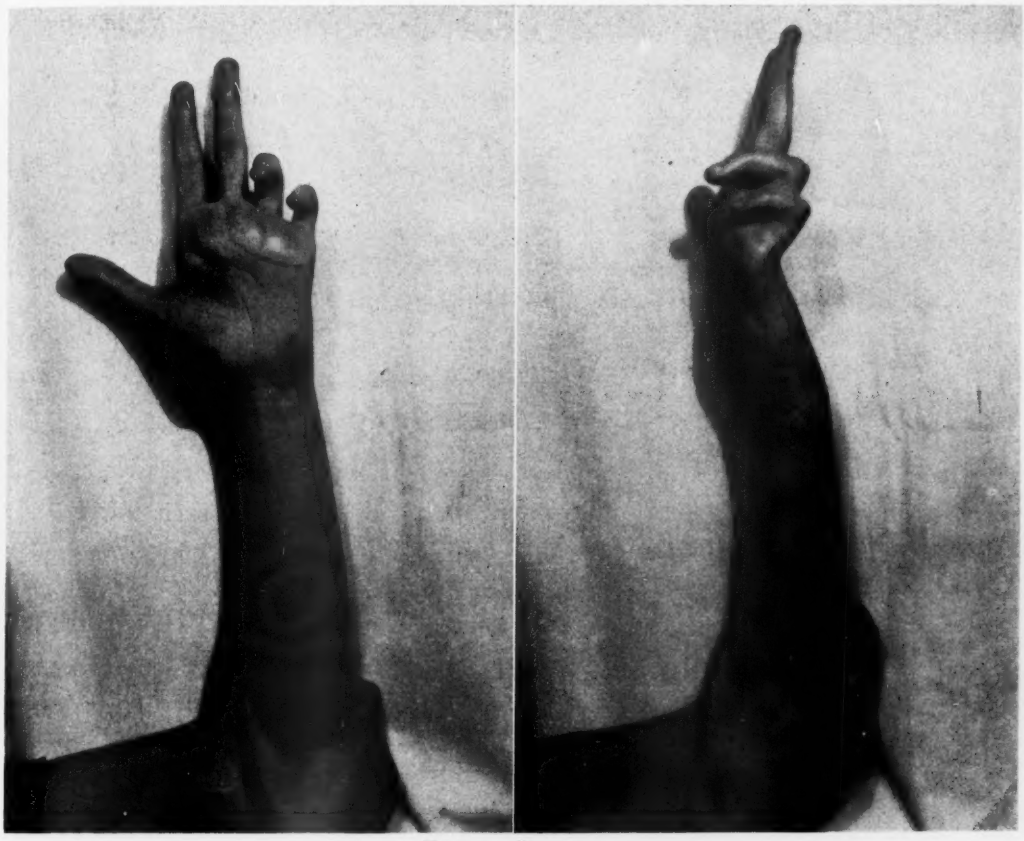


Fig. 15. Case vii.

On September 10, 1936, nerve function was returning and the patient could extend the wrist a few degrees; by December 15, 1936, full nerve recovery was evident (seven months postoperative). After another month the patient was discharged from treatment with elbow extension restricted 35 degrees. End result at one and one-half years postoperative: full nerve recovery, elbow extension restricted 20 degrees.

Case iii. Late repair (seven months) of median nerve at wrist. End result at one year and ten months.

J. H., age 22, single, barman, was injured on May 20, 1935 by a glass cut on the volar aspect of the right wrist, with median nerve severance. He was first seen by us seven months after injury and operated on December 17, 1935. We found a completely severed median nerve, just above annular ligament, with a pseudoneuroma on the proximal end of the nerve. The neuroma was cut back and sutured, flexing the wrist. A cast was in place for forty-five days.

The patient was discharged from the hospital in three days. Six weeks postoperative sinusoidal galvanism was begun, with gradual extension of the wrist. Massage was started eight weeks after operation. In five weeks the patient showed some sensory regeneration to the index and middle fingers and the end of the thumb. He was discharged from treatment August 5, 1936 (seven and one-half months postoperative), after he had resumed work as a barman the month before.

Examination one year postoperative showed full range of digital motion with no muscle atrophy, but the patient claimed some impairment in sensation over the median nerve distribution to the hand and digits.

Case iv. Late repair (thirteen months) of ulnar nerve at elbow.

Mrs. J. E. M., age 35, housewife, was injured

Fig. 16. Case viii.

thirteen months prior to our first examination. She suffered complete ulnar nerve paralysis and extensive scars at the medial and posterior aspects of the elbow and complete flexion contracture of the ring and little fingers. At operation by us July 22, 1937, the ulnar nerve was found severed at the ulnar groove and its ends separated 3 inches, with a neuroma of the proximal end of the nerve. The neuroma was resected, the ends of the nerve sutured, with the wrist and elbow flexed to close the gap. A cast was worn for forty-seven days.

On October 22, 1937 (three months postoperative) there was partial recovery of sensation in the hand and fingers mingled with areas of hyperesthesia. The patient could voluntarily extend the ring and little fingers 20 degrees. Further recovery was expected, but the patient has been unable to come to us for reëxamination as she lives in a distant city. We have heard, however, that she has further improved in sensation and motion.

Case v. Delayed repair (seven weeks) of ulnar nerve at elbow. Later nerve transplant. End result at two years and one month.

A. H., age 42, married, with two children, a milk handler, was injured on July 19, 1935, when he fell from a ladder and cut his left elbow. Some wound infection followed, the wound was sutured by a local doctor. We first examined the patient five weeks after injury and found complete ulnar paralysis distal to the elbow with beginning muscle atrophy, anesthesia and trophic changes.

Operation was performed September 11, 1935, seven weeks after the injury. We found the ulnar nerve severed at the elbow and neuroma formation. The nerve was sutured and passed through the intramuscular tunnel in front of the internal condyle with the elbow flexed. A cast was applied.

Three days after operation the patient left the hospital. The cast was removed forty-two days postoperative, and sinusoidal galvanism was started. The elbow was gradually extended, and sensation was recovered down to the wrist. Treatments were given twice weekly.

The patient returned to work December 9, 1935, three months postoperative. Five months from date of operation we found beginning ulnar causalgia with a painful palpable lump at the point of suture (adhesions). On August 4, 1936 (ten months after the first operation) a

second operation was done, dissecting the scar, freeing perineural adhesions and transplanting the nerve outside the muscles. Causalgia ceased at once.

By October 5, 1936, thirteen months postoperative, there was almost full return of sensation in the hand and fingers, although the little finger remained contracted 25 degrees. The patient used his left hand dumping 40,000 pounds of milk a day from eight-gallon cans. There was an estimated 15 to 20 per cent specific loss in the left hand.

End result on October 26, 1937, two years and one month postoperative, showed almost full sensory recovery and only slight thenar web and interosseous muscle atrophy. The little finger was curled in 45 degree flexion.

Case vi. Immediate suture of ulnar nerve at wrist, and four flexor tendons. End result at three and one-half years.

E. W., age 20, single, a porter, was cut by glass May 17, 1934. Operation was done immediately by us, consuming two and one-half hours. The ulnar nerve and its dorsal cutaneous branch, which were severed, were sutured, and four cut flexor tendons as well. The median nerve was exposed and found to be intact. A cast was applied with wrist flexed.

The patient was discharged from hospital in seven days, but the cast maintained for forty-five days. Then sinusoidal galvanism, heat and massage were started, three times weekly. Ulnar sensory return was first noticed two and one-half months after injury. The patient was discharged from treatment six months postoperative. December 5, 1934, the compensation claim was settled before the Industrial Commission for 20 per cent functional loss in right hand.

The end result at examination on October 16, 1937, three and one-half years after injury showed full ulnar nerve recovery except for slight atrophy of the thenar web and slight restriction in adduction and extension of the ring and little fingers. Full flexor tendon recovery had occurred in the four fingers.

Case vii. Late suture (two and three-quarter years) of ulnar nerve at wrist. End result two years and ten months after operation.

S. V. B., a schoolgirl, age 13, was injured about two years and nine months prior to our first examination on January 3, 1935. A glass cut of the left wrist had left complete ulnar paralysis distal to the scar, with complete contracture of the ring and little fingers. Operation by us on January 5, 1935 showed the ulnar nerve completely severed just proximal to the wrist. The nerve damage was resected and the 1½-inch gap closed by stretching the nerve, flexing the wrist, and suturing the nerve. A cast was worn for fifty-one days, with wrist and elbow flexed.

Sensory recovery was evident beginning thirty-seven days postoperative. Eight weeks postoperative sinusoidal galvanism, heat and massage were started, twice weekly. Three and one-half months postoperative, voluntary motor response was noted in fingers. Six months postoperative, full sensory restoration was present except for dulness to touch and pain at the tip of the little finger. Treatments were then resumed for eleven weeks.

End results two years and ten months after operation: Full sensory return (touch, pain and temperature) in left hand and fingers, but only about 20 per cent recovery of muscle volume and control; some permanent fibrous degeneration and muscle atrophy were noted.

Case viii. Delayed suture of median and ulnar nerves at wrist (seven weeks) and tendon repairs. End result at three years four months.

Mr. R. Z., age 25, married, a truck driver, was injured May 7, 1934, when he fell, cutting his wrist on brass objects. First aid was rendered elsewhere with supposed repair of the flexor tendons. Our first examination three days after injury, showed ulnar and median nerve paralysis distal to the wound, and flexor tendon loss of function to all fingers.

Our operation was done June 29, 1934, seven weeks after injury. The superficial flexor tendons of the middle, ring and little fingers were found sutured to the proximal end of the ulnar nerve, leaving the distal end of the nerve free; median nerve found sutured to superficial flexor tendons of index and middle fingers, distal end of nerve free. There was marked interstitial fibrosis of the median nerve. All structures were repaired, flexing the wrist to close the nerve gap; the flexor carpi ulnaris tendon was transplanted to the superficial flexors of the middle, ring and little fingers because shrinkage of the severed tendons had made their junction impossible. A cast held the elbow and wrist flexed 90 degrees.

The patient was discharged from the hospital in six days. The cast was bivalved in one month and sinusoidal galvanism started, three times

weekly. The cast was removed seven weeks postoperative.

On November 24, 1934, a Volkmann splint was applied to stretch the flexor tendons. At the end of six months ulnar sensation was returning with hyperesthesia. Eleven months after operation median nerve sensation was present in hand and fingers, also ulnar sensation with decreasing hyperesthesia. Thirteen months postoperative the patient returned to work. A month later he was discharged from treatment. Fifteen and one-half months postoperative the claim was settled before the Industrial Commission for 50 per cent specific loss of right hand.

End result three years four months postoperative; full return of median and ulnar nerve function, both sensory and motor, 20 degrees flexion contracture of index, ring and little fingers and 50 degrees contracture of middle finger. 5½ inch operative scar. Some bulging was present in the region of the annular ligament.

Case IX. Late suture (four and one-half months) of sciatic nerve at knee. End result seven and one-half years.

G. V. was injured October, 1930, by a bullet wound behind the left knee. The external and internal popliteal nerves were completely paralyzed. On March 16, 1931, nerve suture was performed by us, five and one-half months after injury. Suture was done under local anesthesia (general anesthesia was contraindicated by a kidney complication).

The peroneal tendons began functioning five and one-half months postoperative. The patient returned to work nine months after operation (he was willing to return to work sooner but his attorney forbade it due to his claim pending settlement).

Examination by us on June 24, 1938, seven and one-half years postoperative, showed: circumference of right calf 14¼ inches; left calf 13¼ inches. The anterior tibial muscles did not function completely, but some anteflexion of foot was produced through the peronei; no Faradic response was present in the anterior tibial group, but strong voluntary action and Faradic response were evolved in the peronei and the posterior group of muscles; the external popliteal nerve suture had resulted very successfully. Sensory return over the anterior tibial distribution was complete (sensation present in the triangle between the great and second toes).

## SUMMARY AND CONCLUSION

Peripheral nerve injuries are discussed in general considerations, diagnosis, detailed technique of nerve suture operations; the after-care, prognosis and results. The more common nerve injuries are described individually, and examples are given in nine case reports of early and delayed nerve suture. A plea is made for early and correct diagnosis, and proper suture and after-care of these important nerve injuries. You can see by these case reports that one can be mistaken very easily in operating on these cases in suturing a tendon to a nerve; but if proper time is taken to study carefully the associated structures, the operator cannot help but see the difference, in that a nerve contains cables of nerve structure where the tendon has a flat surface over cross section. It is very important to study these structures and be sure that you are suturing nerve to nerve and tendon to tendon. These mistakes often cause disability that cannot later be repaired completely.

I wish to express thanks to my associates, Doctors Stimson and Mason, Coleman and Hribel, my photographer, for aid in the preparation of this paper.

# PATHOLOGIC FACTORS FOUND IN THE SURGICAL INVESTIGATION OF EPILEPSY

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THE chronic convulsive state commonly known as epilepsy has so long been considered by neurologists as a cerebral agenesis or degeneration with hereditary tendencies that an attempt to present causative pathologic factors is apt to be received with considerable skepticism, and particularly so when considered surgically. Neurologists do, however, suspect a local cerebral lesion and occasionally invite surgical exploration when attacks are jacksonian in character, or when generalized convulsions are preceded by a definite localizing aura. In the absence of clinical signs of a well-localized cortical irritation, surgical investigation of the epileptic brain has been generally discouraged. This attitude is based on the failure of surgeons to demonstrate a definite pathologic condition which could reasonably be accepted as a causative factor, and on the fact that most surgical procedures have failed to influence the epileptic reaction favorably.

An expanding lesion, or the scar of a localized trauma, may or may not produce sufficient cortical disturbance to induce convulsive phenomena, but, when their occurrence precedes the incidence of attacks, they are usually considered causative, particularly so when attacks exhibit focal symptoms. The great majority of patients presenting clinical phenomena characteristic of epilepsy exhibit no definite focal symptoms. Focal attacks are of great importance from the standpoint of localization when substantiated by neurologic findings but their absence is not sufficient to eliminate even a well-localized lesion, for many brain tumors do not instigate convulsive seizures with jacksonian features, and this is also often true of attacks following local cerebral traumatic lesions.

The "idiopathic" type of epilepsy comprises that great group having periodic epileptic reactions without neurologic evidence of a local cortical irritation, an increase of intracranial pressure, or a definite cerebral traumatic history. They usually present no abnormality demonstrable clinically other than the epileptic reaction, and most patients are apparently perfectly normal between attacks, except that when seizures become frequent and prolonged they usually show signs of mental deterioration. The occurrence of convulsive phenomena (particularly group attacks) in patients who are apparently normal between attacks suggests that there must be some periodic factor active which has escaped recognition. The gliosis so commonly found in the post-mortem examinations of institutional epileptics, i.e., advanced cases, can be attributed to the vascular changes produced by the severe cerebral congestion attending convulsive attacks. There is much evidence to support the assumption that such changes are the result, rather than the cause, of convulsive phenomena.

Convulsive phenomena often attend certain expanding lesions where there is an increase of intracranial pressure; they also occur after cerebral trauma in which there has been a loss of brain substance resulting in an intracranial hypotension. Convulsive phenomena are not dependent on, or even a characteristic reaction of, a general increase of intracranial pressure. Convulsive attacks may develop with the local expansion of a brain tumor, cease after tumor removal, and later recur without a recurrence of the neoplasm. Clinically, the epileptic reactions associated with brain tumor or cerebral trauma do not differ from those seen in the so-called "idiopathic" type of

epilepsy. Even when they are jacksonian in nature, the convulsive reactions of an expanding lesion do not differ from those of a traumatic lesion, and the differentiation between such lesions must be made on grounds other than the observation of attacks.

Thus, we have epileptic reactions which are clinically identical accompanying very diverse pathologic cerebral conditions, such as brain tumor and craniocerebral trauma, in which these lesions may be considered definitely causative. Evidently there exists in these different pathologic states some factor common to both which is capable of producing cortical irritation of sufficient intensity to establish ultimately an epileptogenous zone and the epileptic reaction. This common factor seems to be an increase of local cortical tension. This tension develops in the area surrounding an expanding cortical tumor, in the contraction of a scar, or the tugging on corticodural attachments in traumatic or inflammatory lesions. At operation, particularly under light anesthesia, the traction occurring during the separation of traumatic corticodural adhesions or in the finger enucleation of a tumor will commonly induce convulsive phenomena. This traction element can also be definitely demonstrated in the so-called "idiopathic" type of epilepsy, as we shall see later, where traction may be induced artificially and be followed by typical epileptic reactions. When the traction element is diminished or eliminated, there is a reduction or a complete cessation of epileptic phenomena.

The factors involved in this element of traction in the "idiopathic" type of epilepsy have to do with cerebral postural instability—a disturbance of those factors which normally maintain the brain in postural stability within the cranial box. Through a tear in the arachnoid, fluid leaks into the subdural space; the brain, losing support, gravitates to dependent portions of the skull; traction is exerted on corticodural attachments at the extreme vertex of the brain where the cortical motor centers are located. In time, this traction produces a fairly well-localized area of cortical hyperirritability, an epileptogenous zone, which characterizes lesions responsible for epilepsy and which reacts to stimulation or traction with a convulsion.

In the roentgenologic examination of epileptics, encephalography is commonly used in the hope that visualization of the cerebral ventricles and subarachnoid space may reveal some abnormality. When air is injected into the spinal canal after the removal of cerebrospinal fluid, it ascends into the cerebral ventricles and subarachnoid space. With the introduction of encephalography, it was found that air occasionally escaped into the cerebral subdural space. Investigators of the cerebrospinal fluid pathways—Key and Retzius, Quincke, Weed,[1] and others—have concluded that normally there is no communication between the subarachnoid and subdural space which would allow fluid to escape from the former into the latter. If, after encephalography, air escapes from the subarachnoid into the subdural space, it can only do so through a defect in the arachnoid. If air escapes from the subarachnoid into the subdural space after encephalography, then it is reasonable to expect cerebrospinal fluid to do likewise. I had so commonly encountered great collections of subdural fluid when operating on epileptics for some suspected cortical lesion, that the demonstration of air in the subdural space after encephalography caused me to postulate, some eleven years ago, arachnoidal defects or fistulas through which cerebrospinal fluid entered the subdural space. The exact nature of these fistulas and their location I have recognized only during the past few years, though undoubtedly I had exposed them on many occasions.

*Arachnoidal Fistulas.* Arachnoidal fistulas are defects in the arachnoid through which cerebrospinal fluid leaks into the subdural space. They have thickened edges, and are usually located at the extreme vertex of the brain adjacent to the angle of

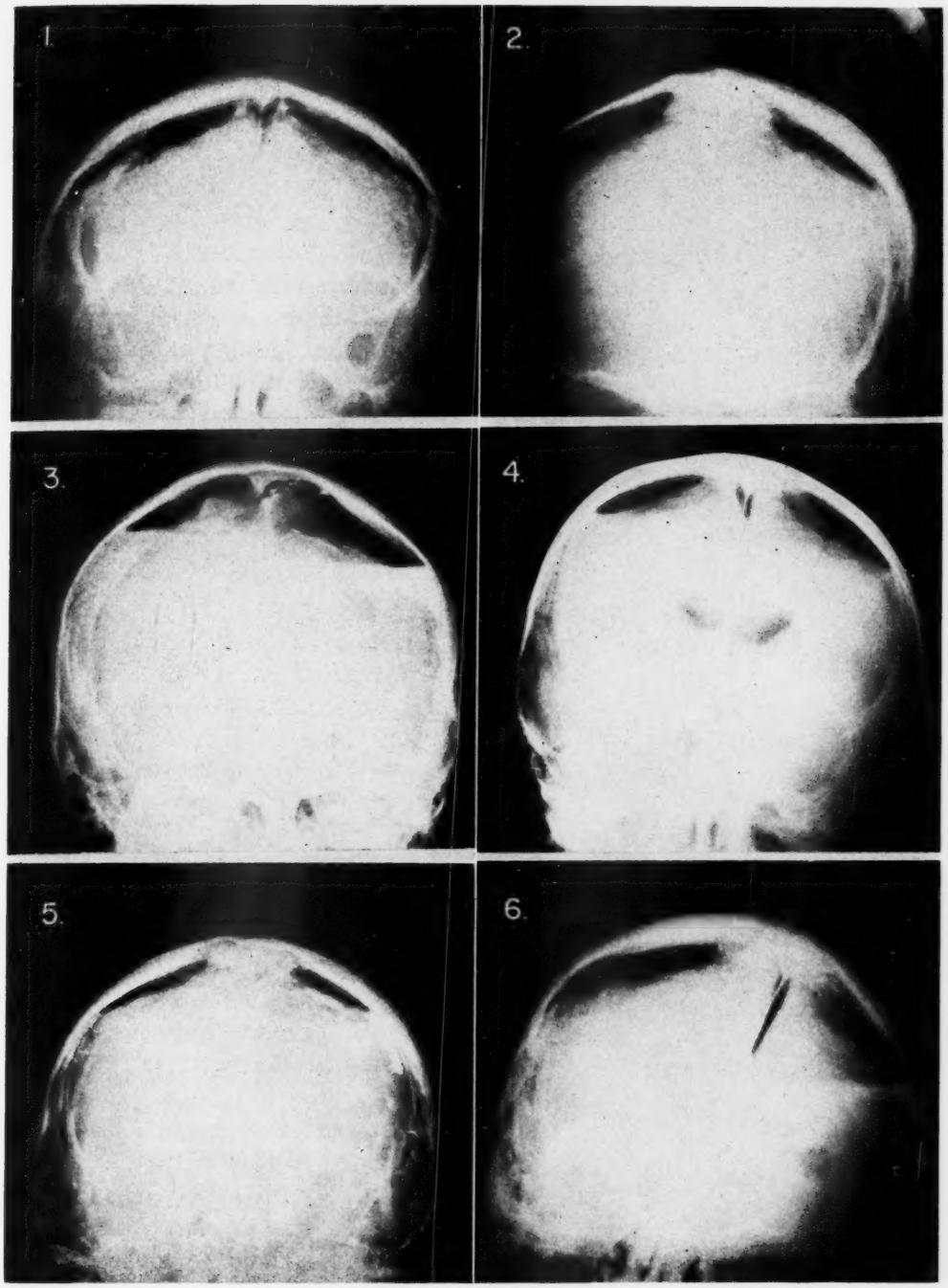


Fig. 1. A series of subdural air insufflations in cases of the so-called "idiopathic" type of epilepsy. 1, shows the slight extent of corticodural attachments adjacent to the sagittal sinus in the frontal region. 2, same patient as 1, showing dense corticodural fixations at the extreme vertex of the skull near the motor area. 3, subdural fluid level demonstrated with floating lipiodol on one side and air, as the contrasting medium, on the other side. 4, extensive corticodural fixations just anterior to the motor area in a child with rather extensive erosion of the skull. The subdural fluid level is shown by contrast with the superimposed air. 5, extensive corticodural fixations just anterior to the motor region with erosion of the skull. Lipiodol used on one side to demonstrate the subdural fluid level. 6, extensive corticodural fixations near the motor area at the vertex. Subdural fluid level demonstrated by a small amount of lipiodol on one side. Lipiodol is no longer used as a contrasting medium for subdural fluid, the superimposed air being sufficient.

a cortical vein as it turns to parallel the sagittal sinus. Since the characteristics of arachnoidal fistulas have been recognized, I have found them at operation in every case of epilepsy. In exposures of the cerebral vertex, when the dura is carefully reflected toward the sagittal sinus, a constant escape of cerebrospinal fluid through these arachnoidal defects is observed. If air is locally injected into the subarachnoid space, it may be directed toward the fistula through which it will escape as bubbles. Apparently these defects are the result of trauma—small tears in the arachnoid, where it is attached to the dura along the sagital sinus, which failed to heal and have persisted as chronic fistulas. Their usual location, at the extreme vertex of the brain where the strain of gravity is greatest in the erect posture, is suggestive as to their origin and persistence.

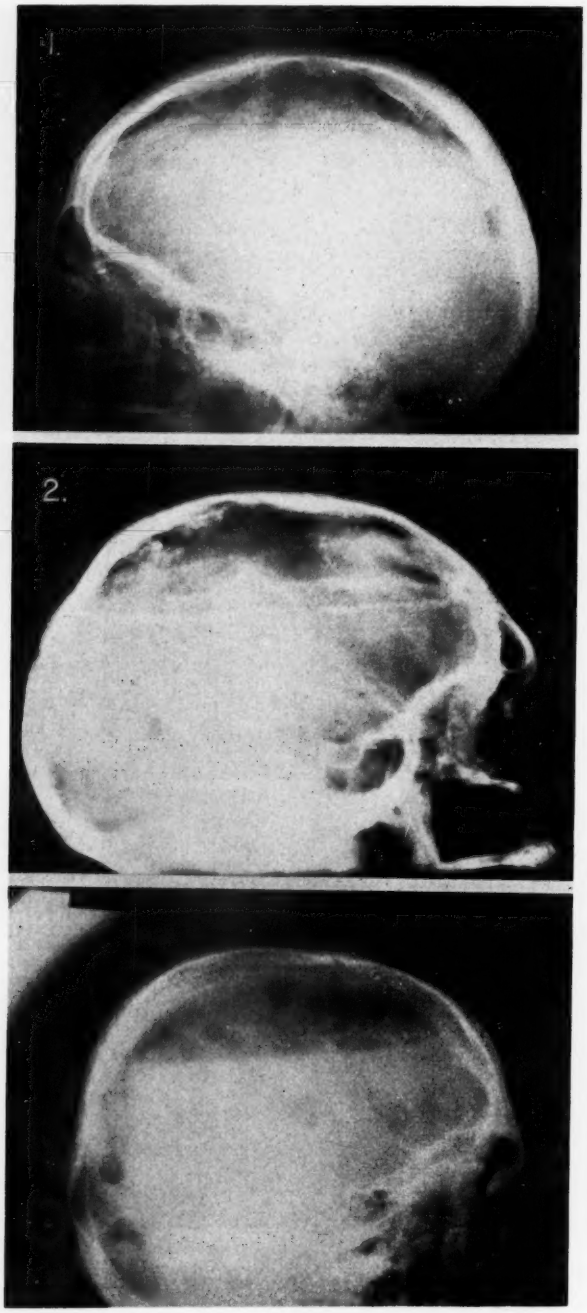


FIG. 2. Subdural air insufflation in typical cases of epilepsy. Lateral views. 1, typical corticodural fixations just posterior to the bregma involving the motor and premotor areas, with some erosion of the skull. 2, corticodural fixations in the motor and premotor areas. Lipiodol floating on the fluid shows clearly a typical fluid level. 3, subdural fluid level contrasted only with air. Corticodural fixations are shown in the motor area. In each of these there will be observed a certain amount of erosion of the bone where corticodural fixations occur.

*Cerebral Postural Stability.* Considering the great weight of the brain, what maintains it in a state of postural stability within the cranial box? The arachnoid is attached at the vertex along the sagittal sinus where the cortical veins turn to parallel the walls of the sinus. There is normally no organic attachment between arachnoid and dura over the lateral surface of the cerebral hemispheres. It has been generally considered that the arachnoid, distended by cerebrospinal fluid under pressure, served to hold the brain in a state of postural stability within the cranial cavity. The arachnoid over the entire surface of the brain sends numerous trabeculae to fuse with the pia, and these hold the brain nicely suspended in the center of the cranial box provided the arachnoid is held in close approximation to the dura. On this arachnoidodural approximation depends the postural stability of the brain within its container. Evidently there are factors other than the pressure of cerebrospinal fluid which maintain arachnoidodural approximation, as it is not disturbed by the withdrawal of cerebrospinal fluid even to the point of producing a negative pressure. Roentgenologic examination after a large quantity of fluid has been withdrawn and partially

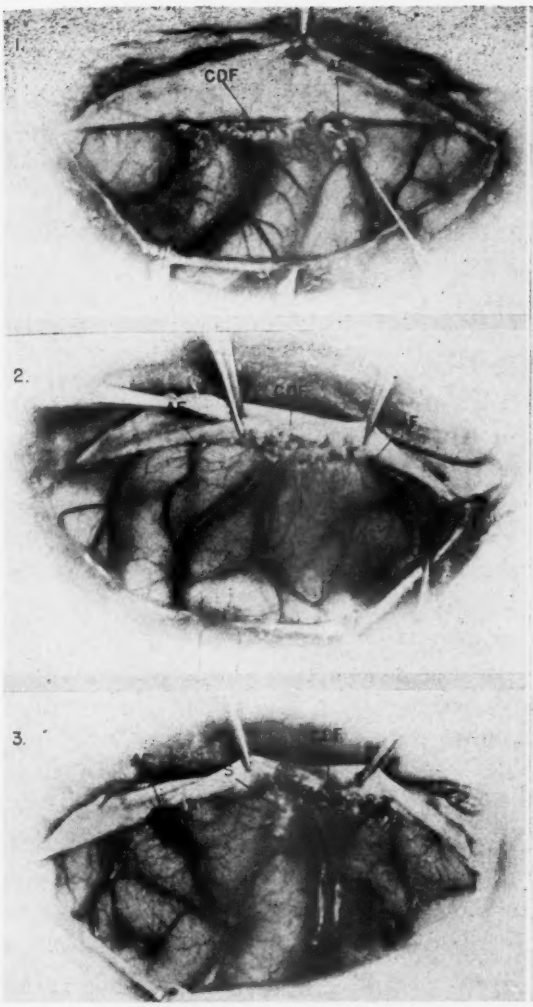


Fig. 3. Series of photographs taken at operation during exposure of the vertex in the surgical treatment of epilepsy. 1, AF, arachnoidal fistula through which bubbles of air are escaping after the local injection of air into the subarachnoid space. CDF, corticodural fixations corresponding to the motor area. Cerebrospinal fluid leaks into the subdural space through arachnoidal fistulas. Postural displacement of the brain occurs, and traction is thrown on corticodural fixations. 2, corticodural fixations in the nature of pacchionian granulations. Two arachnoidal fistulas, AF, are observed on each side of the fixations. 3, CDF, corticodural fixations corresponding to the motor area with partial obstruction of veins. S, scar of arachnoid. AF, typical arachnoidal fistulas.

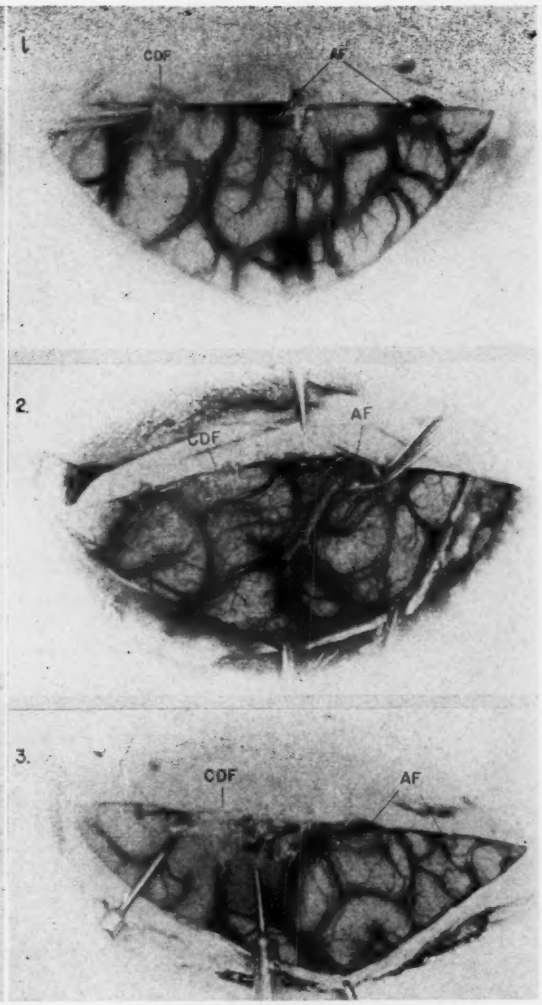


Fig. 4. Series of photographs taken at operation during exposure of the vertex in the surgical treatment of epilepsy. 1, AF, typical arachnoidal fistulas with thickened edges shown as the dura is gently lifted, exposing the cortex adjacent to the sagittal sinus. These fistulas are observed in all cases of epilepsy, and during operation there is a constant escape of cerebrospinal fluid through them. CDF, corticodural fixations with forceps passing under the scarred area which is located just posterior to the motor area. 2, typical corticodural fixations and a typical arachnoidal fistula. Arachnoidal fistula has thickened edges. 3, two arachnoidal fistula, AF. Corticodural fixations, CDF, binding the cortex, arachnoid, and dura in dense adhesions, are observed. The electrodes outline the motor area. Corticodural fixations are surrounded by an epileptogenous zone. Mild faradic stimulation of such an area produces clonic convulsions which pass to the other side of the body and continue after the electrodes are removed.

replaced by air will show the brain in postural stability; if, however, air has entered the subdural space and arachnoido-

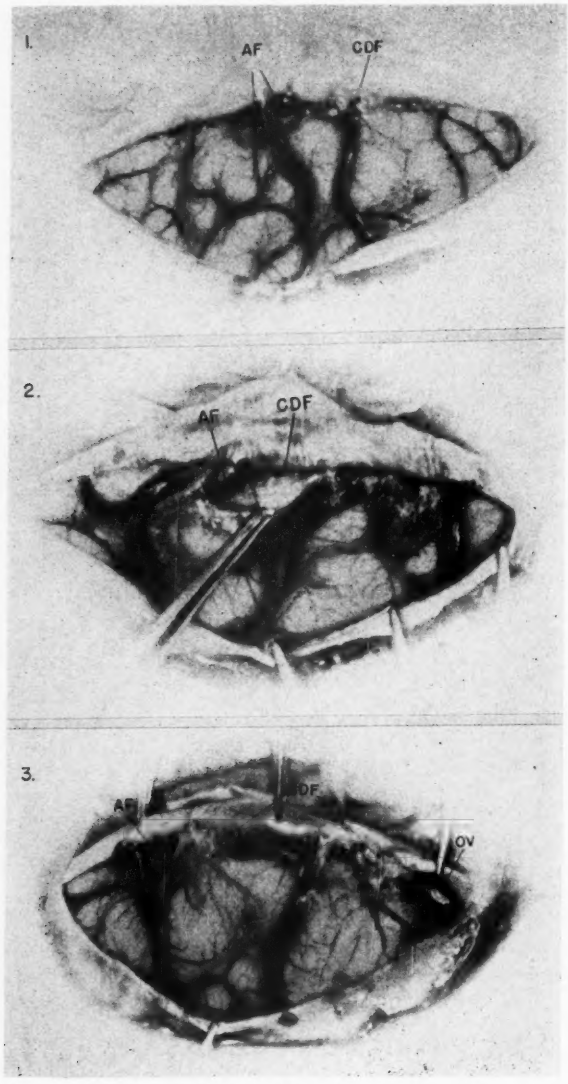


Fig. 5. Series of photographs taken at operation during exposure of the vertex in the surgical treatment of epilepsy. 1, two typical arachnoidal fistulas with thickened, edematous edges. Corticodural fixations are exactly over the motor area. 2, scar infiltrated arachnoid and cortex. Corticodural fixations partially separated, exposing the fistula. Arachnoid beginning to assume a typical jellyfish appearance. 3, a large arachnoidal fistula. Dense corticodural attachments. Obstructed vein, ov, due to corticodural fixations.

dural approximation is lost, cerebral postural displacement will be found to have taken place. *X*-rays show the displaced brain suspended by its attachments along the sagittal sinus, on which, apparently great traction stress is exerted.

The development of subdural air insufflation in the examination of the epileptic brain has made possible the roentgenologic demonstration of corticodural attachments at the vertex; subdural fluid collections; cerebral postural displacement; and actual visualization of the traction on corticodural attachments. It has served also to demonstrate certain factors which maintain cerebral postural stability and which hitherto have apparently remained unrecognized.

*Subdural Air Insufflation.* In 1932, I devised a technique for subdural air insufflation, to be used in the roentgenologic examination of the subdural space for the localization of areas of abnormal corticodural fixations. Subdural air insufflation has become a routine procedure in the examination of the epileptic in my service and has entirely replaced encephalography, although occasionally the two may be combined. The patient, with the back of the head shaved, is seated in a specially devised chair. Through a small burr opening in the occipital region the dura is nicked, and air rushes into the subdural space—it is not injected. For better visualization of the subdural space, from 3 to 5 ounces of cerebrospinal fluid are gradually withdrawn by an assistant while the skull is being perforated. Before the dura is nicked, the lumbar spinal fluid pressure in the sitting posture (normally from 24 to 30 mm. Hg.) is reduced as the spinal fluid is withdrawn. After 1 ounce is removed, the pressure is usually about 14 mm. Hg. After 2 ounces, the pressure is 6 or 4 mm. Hg. By this time the fluid is flowing through the needle very slowly. The dura is now nicked—air rushes into the subdural space, the lumbar pressure immediately rises to approximately 26 mm. Hg. (original pressure), and the cerebrospinal fluid spurts from the needle. This indicates that it is not the pressure of the cerebral fluid that maintains the brain in postural stability within the skull, since postural stability was maintained until air had entered the subdural space.

*Fluid Film Adhesion between Arachnoid and Dura.* Occasionally, during subdural air insufflation, after 1 or 2 ounces of cerebrospinal fluid have been withdrawn by lumbar puncture and its pressure greatly reduced, a nick in the dura (it must be made completely through both layers of the dura) will not be immediately attended by the inrush of air which allows postural displacement of the brain, and the pressure remains very low. After a few deep respirations, however, there is a sudden inrush of air, and the lumbar cerebrospinal fluid pressure rapidly ascends to about its original level. When the dura was nicked, what maintained arachnoidodural contact when the cerebrospinal fluid pressure had been so greatly reduced? There normally

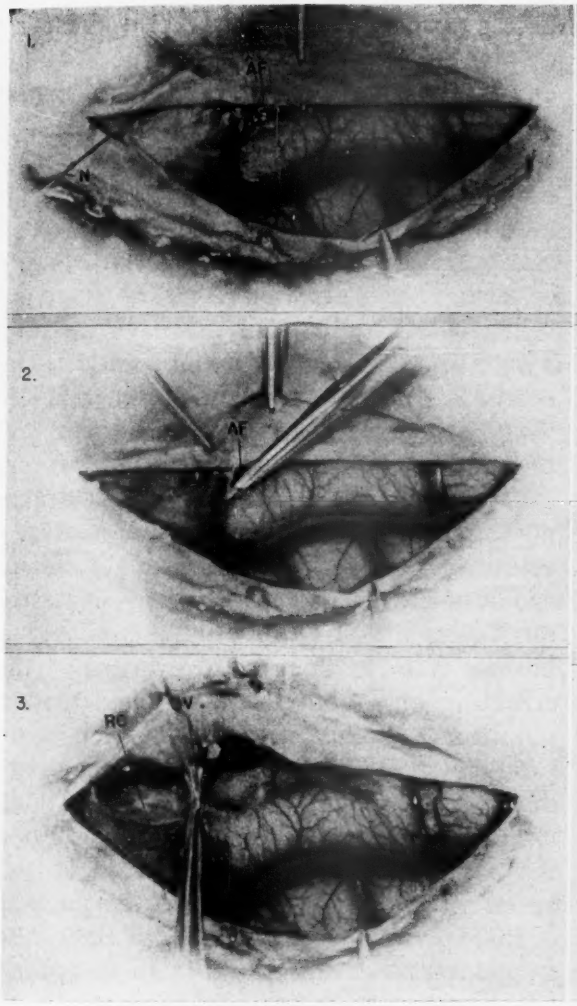


Fig. 6. Series of photographs taken at operation during exposure of the vertex in the surgical treatment of epilepsy. 1, N, local injection of air into the subarachnoid space with the escape of air bubbles through an arachnoidal fistula, AF. 2, forceps passed into the arachnoidal fistula, AF. 3, obstructed vein ligated. Scar obstructing vein has likewise obstructed veins passing along the saggital sinus. RC, retention cyst. Cavity sealed on all sides. Fluid collects in this cavity producing local cortical tension.

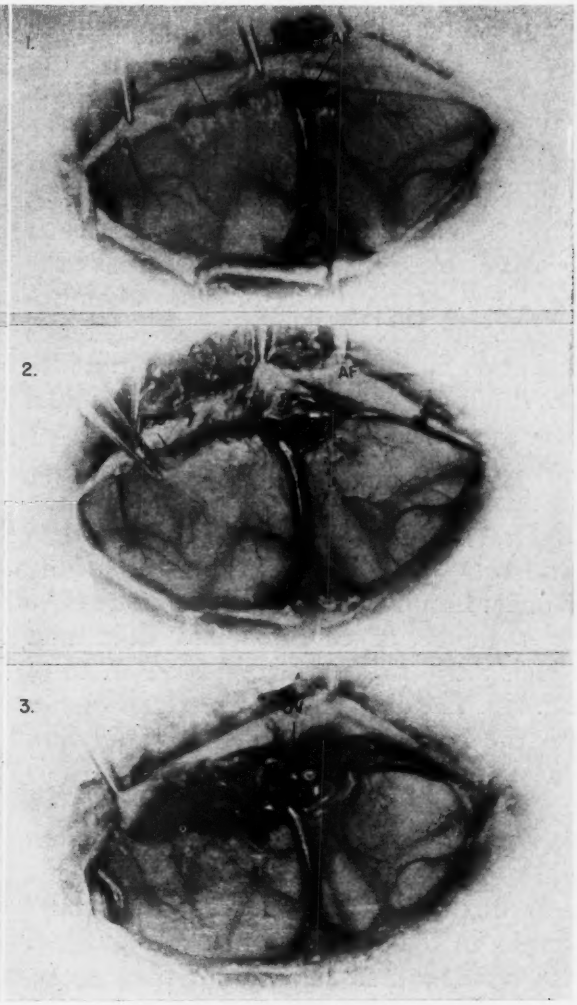


Fig. 7. Series of photographs taken at operation during exposure of the vertex in the surgical treatment of epilepsy. 1, dense corticodural attachments over the motor area with obstruction of veins. 2, area sealed with collection of fluid from a retention cyst. With separation of corticodural attachments, a definite cavity is found containing a number of varicose veins. 3, varicosity of veins paralleling the walls of the sagittal sinus is due to the obstruction of corticodural fixations. Just below OV will be observed the thickened edges of the arachnoidal fistula which loosely surrounds a cortical vein. This patient showed no characteristics of a local lesion. Was classified as, and had been treated for many years for, "idiopathic" epilepsy.

exists between the arachnoid and dura a thin film of fluid which has the adhesive properties common to fluid films. This fluid film adhesion between arachnoid and dura normally maintains them in apposition, and on this cerebral postural stability largely depends.

When fluid film adhesion between the arachnoid and dura is lost, as must occur when there is an excess of subdural fluid, there develops in the subdural space a negative pressure which maintains the brain in a certain degree of postural stability. This negative pressure, however, tends to syphon fluid from the subarachnoid into the subdural space through arachnoidal defects or fistulas, if such exist.

*Subdural Negative Pressure.* A demonstration of the negative pressure of the subdural space may be made during subdural air insufflation in the following manner: Lumbar puncture is done, removing 1 ounce of cerebrospinal fluid and reducing the pressure from, say, 26 to 14 mm. Hg. The dura is now nicked, air rushes into the subdural space as the fluid film adhesion between arachnoid and dura is disturbed, and postural displacement of the brain takes place. The lumber spinal fluid pressure ascends to its original height of approximately 26 mm. Hg. The burr opening in the skull is now firmly plugged with bone wax so that no air may enter. One or 2 ounces more of cerebrospinal fluid are withdrawn, again greatly reducing the lumbar cerebrospinal fluid pressure. The plug in the burr opening of the skull is removed, more air rushes into the subdural space, and the lumbar pressure again ascends with the further postural displacement of the brain. We see then that two factors are responsible for maintenance of the brain in postural stability within the skull: (1) the adhesive action of the normal thin film of subdural fluid which maintains arachnoidodural contact, and (2) a subdural negative pressure which, when film adhesion is disturbed, maintains the brain in a certain degree of postural stability.

Arachnoidal fistulas or defects assume particular importance when these facts related to cerebral postural stability are considered. The presence of an arachnoidal fistula predisposes to cerebral postural displacement, which takes place with an excess of subdural fluid. The maintenance of arachnoidodural film adhesion tends to seal arachnoidal fistulas, but the arachnoidal fistula is a weak point in the arachnoidodural adhesion and contact is easily disturbed. When an excess of fluid collects, cerebral support is then maintained solely by subdural negative pressure. This negative pressure syphons additional cerebrospinal fluid into the subdural space with an increasing degree of cerebral postural displacement. This condition, in which arachnoidal fistulas weaken arachnoidodural film adhesion, constitutes a condition of cerebral postural instability.

The exact outlet by which excessive quantities of subdural fluid escape is not known. We do know, however, that the straining of a convulsion or even that of vomiting, if the strain is sufficient to produce great cerebral congestion, tends to disperse excess subdural fluid, and arachnoidodural contact through film adhesion may then again be reestablished. This is apparently why convulsive seizures partake of their periodic nature, and why attacks so frequently occur in groups. It is not an uncommon occurrence for patients to go one or several months completely free of attacks and be apparently normal, and then suddenly, without apparent reason, have a number of attacks within one or several days. Very often a single convulsive reaction will suffice to remove this subdural fluid factor. Patients who have minor attacks, petit mal, seldom, if ever, have them in groups unless combined with major attacks.

*Corticodural Attachments at the Cerebral Vertex.* In a series comprising some 225 patients having the so-called "idiopathic" type of epilepsy, subdural air insufflation and surgical exposure of the cerebral vertex have been done. In practically all of the

patients in this series there were found at the extreme vertex of the brain, in or adjacent to the cortical motor areas, lateral expansions of corticodural fixations. These corticodural fixations very commonly occur where a large cortical vein approaches the sagittal sinus before turning to parallel the sinus. Occasionally these fixations are in the nature of pacchionian granulations with erosion of the dura and skull. In other cases these pacchionian features are absent but there is a dense corticodural fusion with thick and opaque arachnoid, and, when an attempt is made to separate the dura from the cortex, the pia is usually torn, leaving a denuded area. Very frequently extensive areas of corticodural fixations 2 or 3 cm. wide may be found binding the cortex to the dura and to the walls of the sagittal sinus, and often within these attached areas there are found large varicosities of the cerebral veins which have become obstructed by corticodural fixations as they parallel the walls of the sinus.

The corticodural attachments at the vertex in epilepsy are usually surrounded by a hyperirritable zone which reacts to stimulation with a convulsion. These areas will almost always react to weak faradic stimulation with a convulsion which spreads and continues after the electrodes are removed. If anesthesia is very deep, the convulsive reactions of the epileptogenous zones may be reduced, shortened, or may even be absent. These reactions are quite different from stimulation of the normal motor cortex in which there is a quick tonic reaction resulting usually in a single jerk which is maintained only during stimulation; normal reactions do not spread to other regions unless the stimulation be intense and prolonged, and cease immediately on removal of the electrodes.

Arachnoidal fistulas are very often found adjacent to these lateral expansions of corticodural fixations at the vertex. Occasionally the area around an arachnoidal fistula becomes sealed with adhesions and there forms a cavity into which fluid leaks and is retained. These retention cysts, found in separating corticodural attachments, usually have a soft gray base which is quite different from the scar of arachnoidal infiltration, both grossly and microscopically. Following trauma these corticodural attachments may be located in other regions, but wherever situated their visualization is made possible by subdural air insufflation.

The cortical veins approach the sagittal sinus more or less directly over the cerebral hemispheres, then turn to run forward some distance paralleling the sinus before fusing with its wall. This provision of nature tends to remove the stress on these veins during cerebral postural displacement, but in epilepsy, and possibly in other conditions, large cortical veins are caught in corticodural fixations and anchored so that they are subjected to the full traction stress of a cerebral postural displacement. So commonly do these corticodural fixations occur around large cortical veins at the vertex, that one is inclined to consider the cortical fixations an inflammatory reaction to traction stress, a reaction which ultimately may be so extensive as to obliterate the vein.

*Increased Local Cortical Traction in Epilepsy.* An extremely interesting observation was made by Leyton and Sherrington[2] in 1917: After ablation of portions of the brain in lower apes, collodion dressings were applied to the cortex and convulsions ensued; the removal of the dressings caused their cessation. These experimenters concluded that the convulsions were caused by traction exerted on the cortex with the drying of the collodion dressing. Foerster and Penfield[3] in a paper on traumatic epilepsy called attention to the induction of convulsions during operation by pulling on an adherent dura or by electrical stimulation of the cortex in the region of corticodural attachments. They state, "If the increase of a preëxisting strain produces an attack, it may well be that the preëxisting strain itself is an important factor in the etiology of spontaneous convulsions." Dur-

ing and after the World War I operated on many patients who had had compound craniocerebral injuries resulting in cranial defects which had developed into epilepsy; I found during operation that traction on corticodural attachments would almost invariably incite a convulsive reaction. The freeing of corticodural attachments would result in a cessation of attacks which recurred only if there were a reëstablishment of corticodural fixations.

In brain tumor we have a local increase of cortical tension due to the expansion of the tumor, and some types of tumor, in certain locations, are often accompanied by epileptic reactions. Frequently after such a tumor is removed, convulsive attacks will cease for six months or a year, but with the establishment of corticodural fixations (postsurgical), attacks recur even though there is no recurrence of the tumor—here in one individual we have two forms and two separate occurrences of localized traction with epileptic reactions.* If an increase of local cortical tension or traction is capable of inducing convulsive phenomena in cerebral neoplasms and in traumatic and inflammatory cortical adhesion, one may be justified in considering local cortical traction, if demonstrated in the "idiopathic" type of epilepsy, as an exciting factor. After subdural air insufflation, roentgenologic examination shows very clearly cerebral postural displacement with traction on corticodural attachments at the vertex. Examination, with the patient in the sitting posture and the head tilted at various angles, will reveal a great variation in the width of these attachments in different regions of the vertex. It is not uncommon to find lateral expansions of corticodural fixations extending 2 or 3 cm. from the midline in one location, while immediately anterior or posterior fixations may be very narrow; this local lateral area of corticodural fixations at the vertex is a focus of traction stress during abnormal movements of the brain, and around such a fixed area the cortex becomes hyperirritable—i.e., an epileptogenous zone is formed. The obvious question is, "Are these corticodural fixations abnormal?"—more particularly, "Is traction on them capable of inducing convulsive phenomena?" During subdural air insufflation (without anesthesia) when cerebral postural displacement was artificially induced, 74 per cent of these patients developed convulsive reactions or other type of epileptic manifestations within twenty-four or forty-eight hours, occasionally just after the dura was nicked. Traction, therefore, on these corticodural attachments is capable of instigating attacks.

Subdural air insufflation is accompanied often by headache, nausea, and vomiting, very much as in encephalography but to a lesser degree. The incidence of nausea and vomiting seems to retard convulsive reactions. To mitigate headache and convulsions, which tend to force out the air and subdural fluid, and to insure a more satisfactory roentgenologic examination, patients are usually given from 9 to 12 gr. of phenobarbital in 3 gr. doses immediately before the procedure. About 15 per cent of patients having subdural air insufflation under these relatively large doses of phenobarbital develop convulsive attacks.

## CONCLUSIONS

The presentation of factors apparently active in the production of convulsive phenomena offers certain difficulties in the determination of causative relationship. Only the correction of these abnormalities with a cessation of epileptic phenomena would be the final proof of their causative nature. Two hundred seventy-two patients have been operated on in the endeavor to eliminate traction

* The neurologic mechanism involved in convulsive phenomena I do not pretend to understand, but I feel convinced that it is not directly related to a local vasomotor reaction. I have frequently and carefully examined cortical vessels with a magnifying glass during the induction of a convulsion at operation, and have never observed any evidence of vasoconstriction, either by an increased pallor of the cortex, or in a diametric change in the smaller vessels.

on corticodural attachments at the vertex. Early evaluation of the results has been difficult because of a neurologic mechanism or conditioning process which develops in response to a long continued irritation. The irritation eventually results in an epileptogenous zone, and the epileptic reactions may require many months or years for their development, depending largely on the location of the lesion and intensity of the irritation. I have operated on a number of patients with well-localized cerebral traumas who developed attacks only after six, eight, ten years, or longer. There is good reason to believe that many children developing attacks during the second decade of life do so as a result of an injury sustained at birth. In such instances it is difficult to understand why the mechanism responsible for convulsive reactions was so late in developing, when a definite traumatic causative lesion had apparently existed for many years. It is not my purpose at this time to discuss this mechanism except to say that lesions in certain regions of the brain require a longer period to inaugurate the epileptic reaction. There seems to be a process of conditioning which neurologists look upon as a deteriorating of cerebral functional stability.

The question involved is, if it takes months or years for this epileptic reaction to mature or become conditioned to an irritant, will that mechanism immediately disappear on the removal of the irritation? If attacks should continue, though in diminishing form, after surgical intervention, does this mean that the lesion has been inadequately dealt with? A further complication is that most of these patients have become habituated to large doses of sedative drugs, particularly bromides and barbituates, and the sudden withdrawal of these after they have been used for months or years will usually induce in the epileptic profound psychic or convulsive reactions. We have had this occur repeatedly after operation, have resumed medication, and later have effected a complete but gradual withdrawal of medication with the cessation of attacks. In many instances, after the surgical correction of arachnoidal fistulas, the separation of corticodural attachments, freeing of obstructed cerebral veins, etc., there has been an immediate cessation of attacks; phenobarbital has been gradually eliminated; and patients have remained free from all epileptic manifestations. In some cases more than ten years have elapsed since operation. In others, recovery has been a process of gradual recession in the number and severity of attacks over several years. It would seem that in the latter group time and postoperative medication are necessary for the subsidence of the epileptic mechanism. It suggests that the epileptic zone surrounding a lesion, often requiring years to attain that degree of irritability sufficient to incite the epileptic reaction, will, in some cases, after apparently successful operation (determined by final results), require several years for its subsidence. The fact that certain patients are relieved of their attacks immediately after operation suggests that perhaps in the latter group the lesion was not entirely corrected. Before determining the value of any procedure in the surgical treatment of epilepsy, a number of years must necessarily elapse before results can be evaluated. In this series, in 87 per cent of patients from whom follow-up reports have been received, there has been a complete cessation of attacks or a reduction in the number of attacks by more than 90 per cent.

During the past ten years there have been several variations in the surgical technique used in dealing with traction at the vertex and cerebral postural instability. Analysis of cases grouped according to surgical technique indicates that better results were obtained by some of the methods employed than with others. Since it has been possible to locate arachnoidal fistulas and to deal with them directly, there has been a considerable increase in the number of immediate

or early cessations of attacks, particularly in patients whose onset of attacks had been fairly recent and who had not become habituated to sedatives. Even in the more advanced cases earlier recession of attacks is occurring. This experience suggests that perhaps the subsidence of irritability in the epileptogenous zone might have been more rapid in some of the earlier cases had the present technique then been in use.

This consideration of pathologic factors active in the production of convulsive phenomena in the "idiopathic" type of epilepsy is directed primarily to their consideration as causative agents; it is not my purpose at this time to consider the various surgical procedures used in their correction. That the surgical procedures used have been more or less successful in correcting these factors is obvious when one considers the results obtained. The correction of the factors herein considered has profoundly reduced all types of epileptic manifestations, and this, I believe, definitely indicates their causative nature. The pathologic condition may not always be the same, but, whatever its nature, the factor of local cortical traction seems to be one of great importance in the induction of convulsive phenomena.

While local cortical traction may be considered the exciting factor in the chronic convulsive state, the traction is made possible through those factors which are responsible for the cerebral postural instability, that is, the loss of fluid film adhesion between arachnoid and dura due to arachnoidal fistulas. The traction becomes intensified in local areas of the cortex which have undergone fixations to the dura, and it is quite possible that these corticodural fixations are due to the traction stress thrown on cortical veins at the vertex, which induces an inflammatory reaction with scar infiltration of arachnoid and pia. These fixations in some instances are undoubtedly due to trauma, possibly the tearing of veins passing through the subdural space resulting in collections of subdural fluid and blood. The principal rôle of trauma, however, lies in arachnoidal tears which, failing to heal, become permanent fistulas through which fluid periodically leaks into the subdural space.

## SUMMARY

1. In a series of 272 patients with the so-called "idiopathic" type of epilepsy, certain definite lesions have almost invariably been found at operation, the more common types being shown photographically.

2. In some 225 of these cases, corticodural fixations at the cerebral vertex in or near the cortical motor areas were localized roentgenologically by subdural air insufflation, in which cerebral postural displacement was artificially induced by allowing air to enter the subdural space.

3. Arachnoidal defects or fistulas through which cerebrospinal fluid leaks into the subdural space have been found in practically every epileptic patient operated on since their characteristics have been recognized. These fistulas are probably the result of tears in the arachnoid which have failed to heal. They are usually located at the extreme vertex of the brain where the strain of gravity is greatest.

4. During subdural air insufflation it is possible to demonstrate that cerebral postural stability is dependent on the maintenance of arachnoidodural approximation. This approximation is controlled by two factors: (1) fluid film adhesion between arachnoid and dura which maintains these structures in forceful apposition; (2) a negative subdural pressure which develops when fluid film adhesion is lost through an excess of subdural fluid. This subdural negative pressure maintains the brain in a certain degree of postural stability but tends to syphon cerebrospinal fluid into the subdural space through arachnoidal fistulas when such are present.

5. The traction on the cortex which occurs in expansive lesions and corticodural fixations after craniocerebral trauma seems

to be a factor responsible for the induction of convulsive phenomena in these two diverse pathologic conditions. During operation traction will serve to induce attacks. In the "idiopathic" type of epilepsy definite traction is exerted on corticodural attachments at the extreme vertex of the brain during postural displacement which occurs when there is an excess of subdural fluid. Subdural air insufflation, devised for the examination of the subdural space, serves in the localization of corticodural attachments; it shows the cerebral postural displacement which takes place when air or excessive fluid enters the subdural space; subdural fluid is determined by a definite fluid level which changes with position of the head.

6. In this series the surgical correction of arachnoidal fistulas and the reduction of traction on corticodural attachments have so diminished convulsive attacks in the epileptic that one is justified in considering the lesions which are herein presented as definite causative factors in the chronic convulsive state.

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Treatment [of septic wounds] has radically altered since the days of the war of 1914–18. The advocates of irrigation now recognize that "it is the mechanical washing far more than the action of the antiseptic that is responsible for any improvement." . . . I doubt if irrigation will long survive at all.

From—"War Wounds and Air Raid Casualties" (Lewis).

# THE RELATION OF CERTAIN SEMINAL FINDINGS TO FERTILITY, WITH SPECIAL REFERENCE TO SPERM CONCENTRATION AND THE SIGNIFICANCE OF TESTICULAR EPITHELIAL CELLS IN SEMEN

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IT is axiomatic in nature that the soil determines the crop, both as regards quantity and quality. In the same way testicular function is evidenced by the seminal picture. In 1936 I reviewed before the New York Obstetrical Society the results of the morphologic and biometric studies of the semen during the previous ten years.[30] I naturally could not cover the entire subject because of lack of time. Thus I could not go into the subject of sperm concentration, nor consider the significance of certain epithelial cell forms found in the seminal fluid. These cells I have studied especially during the last three years.

I have pointed out repeatedly that most of the reports on the relation of oligospermia to fertility were of no value because they failed to take into consideration the morphologic and biometric characteristics of the spermatozoa and thus did not separate oligospermia with normal sperm from oligospermia in which the poor quality of the sperms would in itself account for the infertility present.

## I. THE RELATION OF THE NUMBER OF SPERMS TO FERTILITY

The opinions regarding the number of sperms necessary for fertility vary tremendously. Thus the normal number of sperms per c.c. for man is believed by many to lie around 100 million whereas other authors accept a much lower figure. I have examined and counted many semen specimens and 100 millions per c.c. seems to me to be too high as an average. With a Leitz microscope, a number 6 objective and a 10X ocular and the usual tube length (140 mm.) the approximate area of the visible field is $\frac{1}{10}$ square mm. If a thick drop of semen is used and a thin cover glass the approximate thickness of the seminal layer will be 0.1 mm. A short calculation will show that one sperm to a field will equal 100 sperms per c. mm. or 100,000 per c.c. Where 100 million sperms per c.c. are present, each microscopic field would show 1000 sperms, certainly a picture beyond the average.

The above described method of determining the number of sperms per c.c. is accurate enough for all general purposes. Even the results of instituted treatments can be so evaluated since normally semen specimens from the same individual under apparently identical conditions vary quite considerably. For accurate work, of course, a counting chamber must be used (hemocytometer). As many fields as possible should be counted (I count all the 400 small squares) since an even distribution of the sperms is not easy to attain.

It is well in all cases (except where very few sperms are present) to dilute the semen, since its density may otherwise make a bothersome focussing up and down necessary to see the sperms at the various levels of the 0.1 mm. deep counting chamber. I personally prefer a 1 per cent chlorozene solution as a diluent since it kills the sperms and also dissolves most of the mucus present. Specimens of semen with good sperm concentration have to be diluted and the dilution must vary according to the sperm concentration present in the particular specimen. A dilution in the gross is much preferable and more accurate than pipette dilution. If desired a coloring

agent such as methylene blue or cresyl violet may be added.

In thus counting the number of spermatozoa per c.c. what may we interpret as oligospermia of sufficient degree to influence fertility? Certainly, as many authors have pointed out, as I have for man[10] and Polozow[33] and Polozow and Nagajew[34] for the stallion, the capacity for the production of spermatozoa varies very much in different individuals without there being any evidence of disturbed fertility. I feel sure that Macomber and Saunders[35] have disregarded other factors which might have caused infertility when they claimed that fertility was practically never present with sperm concentrations of less than 60 million per c.c.

The same authors believe that it is the sperm concentration and not the total number of sperms present in the ejaculate which is the important factor. While the importance of sperm concentration cannot be denied (Cohn,[36] Fuchs[37]) the total number of the spermatozoa seems to be of far greater importance. Hutt,[38] for example, has shown for the rooster that high sperm concentrations do not mean high fertility. The least fertile of his roosters had twice as many sperms per c.c. as the one with the highest fertility, and Hutt states: "Fertility in the male fowl is dependent upon physiological efficiency of the spermatozoa rather than upon their quantitative production."

Lloyd Jones and Hays[39] found that in a male rabbit twenty services in four hours decreased the number of sperms from 35.6 millions per c.c. to 300,000 per c.c. and the fertility from 72.09 per cent to 35.55 per cent. But the fact that even with the latter figure conception still occurred in about one-third of the cases shows that here again nature is very lavish and allows very great reduction from the optimum before she admits defeat.

Walton,[40] taking sperm from the epididymis of rabbits, found that fertility was definitely reduced by dilution of the semen with normal saline to a concentration of one million or less and that sterility was present when the sperms averaged less than 10,000 per c.c. It is to be noted, furthermore, that in these experiments the spermatozoa were taken from the epididymis and so probably were not fully matured. In addition, the diluent changed environment, buffer reaction, osmotic tension, etc.

Iwanow, Kusnetzowa, Milowanow, Neumann, Nagajew, Skatkin[41] used bull sperm, diluted thirty-two times, for artificial insemination with very good results. These authors state that they feel that even a much greater dilution could have been used satisfactorily.

Lagerloef[42] in his exhaustive monograph on the sperm morphology of the bull, which he compared directly with the pathologic findings in the testes in many cases, says: "My investigations show that the usual cause of decreased sperm production is a degeneration of the seminal tubule epithelium. Whatever the agent is which produces these changes it causes in most cases a disturbance of spermatogenesis as a whole and not only a decrease of the number of sperms."

We see then that a very great reduction of the number of sperms can occur before actual sterility supervenes. My own cases fully corroborate this. I will cite here only two of them.

Case 1. M. M., a white male, 40 years old, married five years. His wife, 30 years old, was a hypothyroid. No contraceptives had ever been used. Three spontaneous abortions had occurred at three to four months of gestation, the last one two and one-half months before examination.

Semen specimen was about 2.5 c.c. in amount, slightly increased in viscosity. Motility was good and continuous for many hours in about half of the sperms. The total number of the sperms was 1,800,000 per c.c. Sperm morphology was slightly above the normal limit: 24 per cent head changes, made up of 9 per cent small heads, 1 per cent large, 1 per cent tapering, 9 per cent round and 4 per cent miscellaneous. Since round heads are in most cases not of great significance I feel that probably the hypothyroidism of the wife

played a role in the abortions. Other endocrine imbalances may also be important but the wife so far has not been willing to have the proper examinations carried out.

I have emphasized before[14,30] the difficulties of determining motility of the spermatozoa accurately; it is not necessary to go into this question here. I do, however, want to state that I believe the determination of the percentage of motile sperm in any specimen to be a very haphazard procedure. Often one sees a sperm, characterized by some peculiarity actively motile, and the same cell absolutely immotile a few minutes later and later again we find the same cell with good motility. What is true of one sperm is of course true of others also. Some semen specimens from known fertile men, because of the effect of some almost imponderable agent or agents, will show only a relatively small number of motile spermatozoa.

The second case may illustrate this point as well as definite oligospermia with normal clinical fertility.

Case II. G. M. Semen (friction specimen in office) 2.5 c.c. in amount was grossly normal, with one million sperms to the c.c. Very active motility of apparently less than half of the sperms was present. Morphology and biometrics were normal. The clinical fertility of this man was very high.

I agree with Macomber and Saunders[35] that small testes usually produce fewer sperms than large normal ones, but I cannot agree with them in believing that nature will function only in such narrow limits as are represented by the difference between 60 and 100 or more millions. I subscribe to the opinion of Hutt[38] that fertility depends not on the quantitative production of the sperms but on the physiologic efficiency of those produced.

I still feel as I did years ago that if twenty-five to fifty otherwise normal sperms are present in each high dry power microscopic field, this number should be sufficient to insure offspring, at least under the conditions existing among humans. I want to emphasize, however, that motility does not indicate the fertilizing power of the spermatozoon.

Perhaps some readers have also expected here a consideration of the hydrogen ion concentration of the semen. However, as this is usually carried out, it is of no value, and investigations by many workers have shown that the buffer reaction of the seminal fluid is such that in most cases the sperms are well protected for many hours and minor variations of the hydrogen ion concentration mean little or nothing.

## II. Some Notes on Sperm Morphology Especially on the Significance of Certain Cell Forms Found in the Semen

In previous reports I have discussed and pictured various sperm abnormalities to the number of about fifty. Some authors since then, especially Belding,[43] Generales and Stiasny[44] have gone much further and have divided the various morphologic sperm forms into hundreds of types. This to me seems an unnecessary complication of details. It is self evident that one can divide the large sperm heads into large round, narrow, tapering, irregular, and add differences in staining reactions. Any one of the other main types of sperm head forms can also be thus split up into subtypes. The result will be the tabulation of an enormous number of different forms which will, however, not lead to any clarification of the subject. On the contrary, it will only lead to confusion.

In the morphological evaluation of the spermatozoa I have always advised that one take the most prominent feature of the sperm and sperm head as the criterion, as for example, large, small, narrow, etc. If we divide all our groups into subgroups the result will be not only that the fine differences will make it impossible to agree with ourselves in recounting the same specimen but the individual groups will be so small that their proper meaning will be next to impossible to determine. Aside from this,

there is great danger that, looking for too fine distinctions will cause variations which should still be considered normal to be tabulated as abnormal. The difficulty in evaluating any semen specimen as to its fertilizing powers lies not in picking out definitely narrow, large, small cells, etc., but in knowing where to draw the line between normal and abnormal.*

For the average report of the morphologic characteristics of a semen sample I use only the classifications: large, small, narrow, tapering, round and miscellaneous head forms. The last group includes thickened end knobs, aberrations of the staining reactions, amorphous forms, etc. The body changes are classified as thickened body, coiled, double, and miscellaneous—which last group includes such forms as abaxial implantation, naked body fibril, etc. Usually the cell forms grouped under miscellaneous are so few in number that reporting them on a percentage basis would give a false impression. If, for instance, one sperm in 500 is found with an abaxial implantation one cannot say that $\frac{1}{5}$ per cent of the sperms are abaxial because the next thousand cells or more may not show a similar change. Should, however, one of the sperm forms listed under miscellaneous be quite frequent in a semen specimen, then such forms are taken out of the miscellaneous group, placed under a specific heading, and specially noted, just as the presence of certain immature forms and cells of spermatogenesis are separately indexed. Of these forms I will speak later.

I do feel, however, that while in the classification cited, all narrow and all tapering sperm heads should be grouped together one should in addition distinguish between long and short narrow and tapering forms. (Fig. 1.) I have always stressed the sinister import of these narrow and tapering sperm heads and have made special notes on their presence, especially of the very long heads and the sickle-shaped ones. (Figs. 2, 3 and 4.) The proof that these head changes are really very important has been given by Lagerloef[42] in the bull. This author examined both semen and testes of bulls and found that tapering and what he calls pear-shaped heads are especially frequent in severe types of testicular degeneration.

Aside from the sperms themselves there are, however, other cell elements present in the semen. To demonstrate them the cleared smears used for sperm morphology are of no value. I use thin smears quickly dried in a current of air and stained without fixing if the stain used contains alcohol. If it does not then 50 per cent and 95 per cent alcohol is used successively as a fixative. Good results can be obtained with an old (polychrome) methylene blue, or with eosin and methylene blue. The carbol-fuchsin-eosin stain I usually employ for the spermatozoa may also be used.

The non-spermal cells of the semen can be grouped under three headings:

1. Red and white blood cells. Such cells may very occasionally be found in normal semen, but when present in greater numbers red blood cells and leucocytes, especially in clumps, denote congestion and inflammation. The approximate site of such an inflammation may at times be diagnosed by the type of the other cell elements present, such as urethral, prostatic or testicular cells. Since the meaning of red blood cells and white blood cells as well as

* My own investigations and those of others so far seem to show that a proper evaluation of the sperm morphology and biometrics in all their aspects allows conclusions to be drawn about the fertility of the respective individual. My own attempts to disprove this hypothesis have always been unsuccessful. In *J. A. M. A.*, 112: 1817, 1939, Frances I. Seymour published a report of a case in which only 3 per cent of the sperm heads were abnormal in a semen specimen containing 94,000,000 sperms per c.c. of good motility. No mention is made of the biometric findings and perhaps these were abnormal. Morphology and biometrics usually run parallel, but this is not necessarily so, as I have pointed out before. I have never seen any case with only 3 per cent abnormal head forms among hundreds of normal seminal specimens with good clinical fertility. Even a normal bull, where the head changes are less frequent than in man, generally has more than 3 per cent abnormal head forms. I seriously think that the morphological count must have been made a little too liberally. Arrangements have been made to check this case and it will be reported upon later because of the importance of the question to be settled.

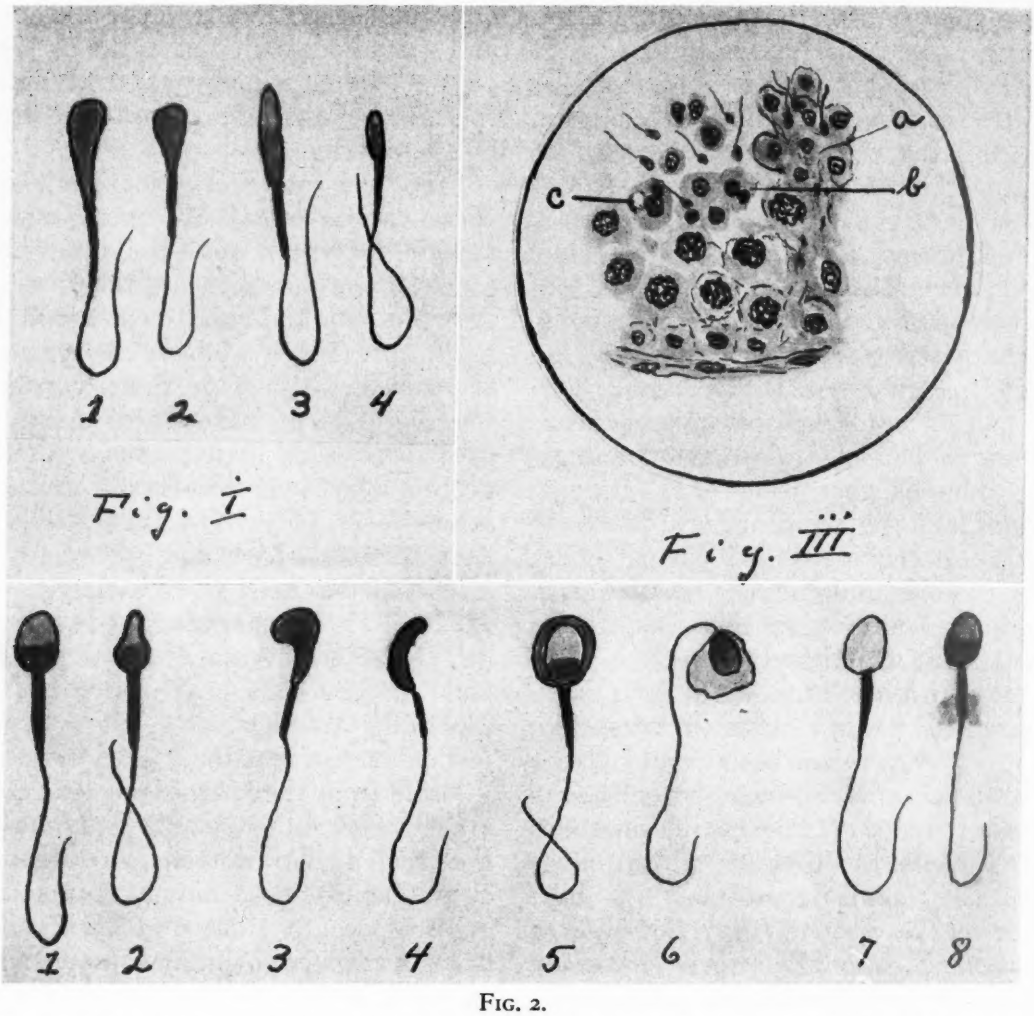


Fig. 2.

Fig. 1. Cells 1 and 2 represent tapering sperm heads and cells 3 and 4 narrow sperm heads. Sperms 2 and 4 are the usual forms and 1 and 3 very long types which are especially indicative of severe spermatogenic disturbance. Often the first sign of improvement in a semen specimen is a shortening of these very long forms, resulting of course in a decreased value of the coefficient of variability while the morphological cell count still remains the same. (See text.)

Fig. 2. Sperms 1 and 2 are normal forms (cell 2 seen from the side). Sperms 3 and 4 are sickle-shaped heads, like the tapering and narrow forms, of definitely sinister import. Sperm 5 still has a thick mass of granular cytoplasm (spermatid rest) around the head. This cytoplasm stained much darker than normal. Sperm 6 also has an irregular cytoplasmic mass around the head. There is a tail but no body present. Sperm 7 is an almost unstained cell head. I believe these sperms are old and degenerating forms which are being cast off. They seem to be more frequent after prolonged periods of sexual rest. Sperm 8 is normal except for some mucus adhering to the body. I believe this to be of no significance.

Fig. 3. Portion of seminiferous tubule of a human, adult testicle. From below upwards we see the tubule wall with light staining, elongated nuclei. Then a single layer of spermatogonia. Above these are two layers of spermatocytes with large nuclei with the chromatin in a skein-like formation. Above the spermatocytes are the spermatids, smaller cells which form the spermatozoa. *a*, a sertolian cell. This with the attached spermatids and the embedded spermatozoa forms the so called spermatoblast. *b* and *c*, spermatids with two nuclei. The one at *c* also shows a vacuole. (Drawn semischematically at a magnification of 400 diameters.)

their cytology is well known it is not necessary to discuss them further.

2. Epithelial cells from the male tubular system, tubuli recti, vas deferens, prostate, urethra or urinary tract itself. Here we find columnar, transitional and squamous epithelial cells. Their cytology also is well known and needs no discussion here. Any one of these cell forms may be found singly in normal semen but when present in greater numbers or in connected sheets they naturally denote irritation of some sort. It might be well here to mention also the corpora amylacea of the prostate, laminated in appearance and staining with iodine. These bodies are found especially in exhaustion and with increasing old age when they may form concretions up to 1 mm. or so in diameter. Usually they are 10 to 30 microns in size.

3. Cells from the testes themselves (seminal tubule epithelium). Some of these cells, for instance spermatids, are occasionally found in normal semen ejaculates but their numbers and types are much increased in abnormal cases. All these cell forms have been described years ago by Broman,[45] Stieve,[46] and others, but it is their diagnostic significance in semen evaluation that I wish to stress here. In order to understand this significance it is necessary to review briefly the spermatogenetic processes.

The seminiferous tubules, invested with a thin connective tissue membrane, show a very characteristic epithelial lining which can be divided into three different groups of cells. (Fig. 3.) On the basement membrane lies a single layer of rather small cuboidal spermatogonia. These divide to form spermatogonia and spermatocytes of the first order, rather large cells with very large nuclei and with a skein-like arrangement of the chromatin. From these the spermatocytes of the second order are formed and these in turn form the spermatids, small spherical cells with little cytoplasm and a large spherical nucleus showing irregular karyosomes. In this process the spermatocytes of the second order each form two spermatids, presumably by amitotic division, so that the number of chromosomes is reduced to one-

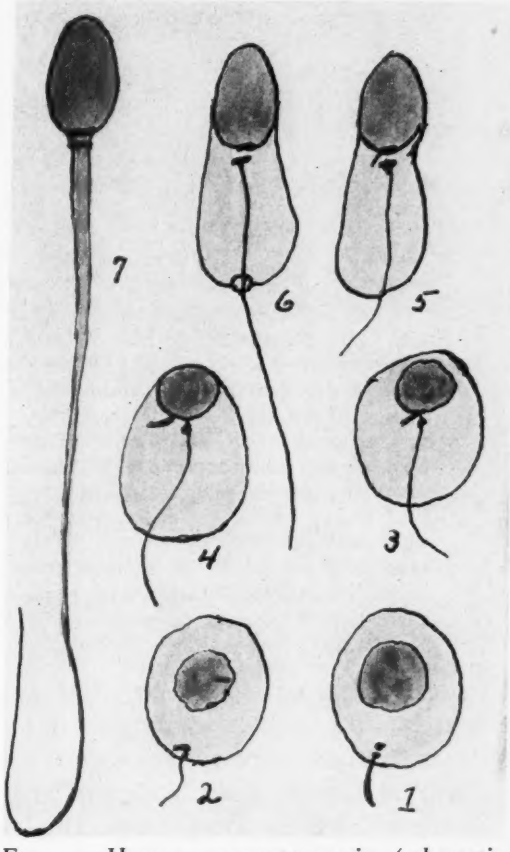


Fig. 4. Human spermatogenesis (schematic according to Meves, Bonnet). The division of the centrosome, formation of the end knobs (later in the base of the sperm head), tail and end ring are shown. Also the gradual protrusion of the sperm head from the cytoplasm of the original spermatid. (Compare with Fig. 6.)

half of the original number. The spermatids then change to spermatozoa. (Fig. 4.)

It is only in the spermatid that the centrosome divides and a tail grows from the posterior half. Such cells as Carey and Hotchkiss[47] present as spermatocytes are therefore not spermatocytes but abnormally developing spermatids of the type I have pictured before. (I might add here also, not in a spirit of criticism but simply to avoid someone's being led astray, that I cannot agree with them on their Figure 1 which they label normal spermatozoa. If the top cell is normal then the lowest one is certainly too small, or vice versa. The

central sperm is long and narrow, with a definite taper and seems to have an abaxial implantation.)

double nuclei and with vacuoles are seen. (Fig. 3.)

Spermatid giant cells, evidently caused

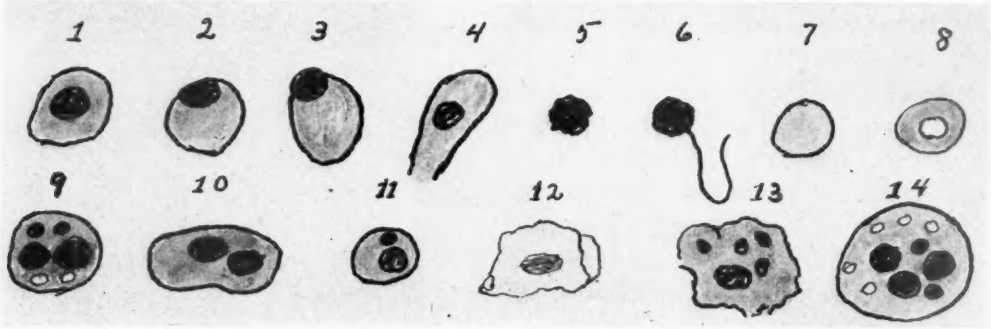


Fig. 5. Cell 1 is a spermatid with a rather excessive amount of cytoplasm. Cells 2 and 3 are spermatids with the nucleus at the periphery and protruding. Number 5 is an extruded spermatid nucleus, number 6 one with rudimentary tail. (Compare with number 1 in Fig. 6.) Cell 4 may be part of the sertolian cell system, though probably it is a deformed spermatid. Number 7 is a protoplasmic drop. Such drops are frequently seen in sizes from 2 to 10 and more microns. Cell 8 is a degenerating spermatid, the nucleus of which remains practically unstained. Cell 9 is a giant multinuclear spermatid with vacuolization. Cell 10 is similar to 9 but has no vacuoles. Cell 11 is a normal sized binuclear spermatid. Cell 12 a squamous epithelium cell from the urethra or skin. Cell 13 apparently has been torn loose from a spermatoblast, for it contains several sperm heads. These cells some authors have interpreted as macrophagocytes but Stieve[46] believes that they are the result of cell fusion "sperm agglutination." I believe in some cases similar pictures are produced by active penetration of the spermatozoa into the cell. (See text.) Cell 14 is a huge, multinuclear, degenerating spermatid with vacuoles.

Besides the spermatogonia, the spermatocytes and the spermatids in the testicular tubules there are also present the so-called "sertolian" cells, large and irregular in form, which arise from the primordial cells. They grow upward like bushes, unite with the spermatids and are the embedding medium for the developing spermatozoa which break away when their development is completed. The union of the sertolian cell and the spermatid forms the "spermatoblast."

Since testicular epithelium is very sensitive, aberrations from the described and pictured development often occur. In fact Kyrle,[48] Schinz and Slotopolsky,[49] Romeis[50] and others have always found in boys and adult men evidence of atrophy in some of the seminiferous tubules. They consider such localized atrophy as part of the normal picture. Although Stieve[46] denies this, I myself have generally seen some tubules in an apparently normal human testicle which showed no spermatogenesis and at times some degeneration (perhaps temporary). Occasionally spermatids with by fusion, are at times found and although these cells of course indicate degeneration, the rest of the slide may seem normal. When such cells lie loose in the lumen of the testicular tubule, as they frequently do, they of course may be expelled with the semen. However, Lagerloef[42] states that while he has seen such cell forms in the epididymis and even in ejaculated bull semen, he has never seen spermatocytes or spermatogonia there. This in general has been my experience also, although spermatids are not infrequently found in both normal and abnormal semen specimens. It is true, however, that most of these show some more or less marked evidence of degeneration.

Cell 1 of Figure 5, for example, seems too large; and cell forms such as 2 and 3 are certainly not normal. At this stage, when the nucleus is at the periphery, a comparison with Figure 4 shows that evidence of tail formation should be present. Sometimes such forms as 5 and 6 are also seen in the semen. Number 5 is evidently an extruded spermatid nucleus and number 6

even has a rudimentary tail but no body (compare with number 1 in Figure 6). Many semen specimens also show vesicular

With one exception, to be described later, I have, like Lagerloef[42] never seen spermatocytes or spermatogonia in human

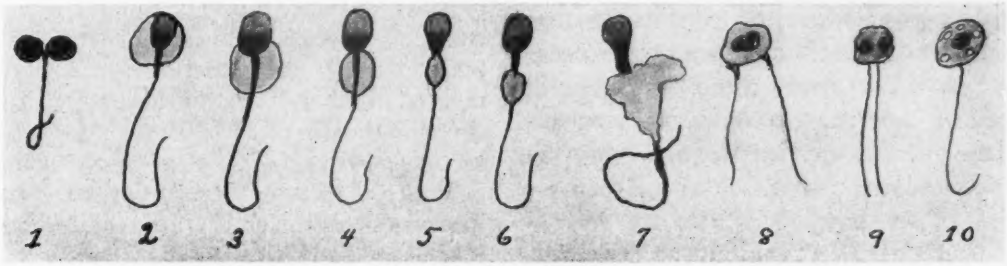


Fig. 6. Number 1 is an extrusion of the nuclei of a binuclear spermatid. There is a rudimentary tail but no body present. Sperms 2, 3, 4, 5 and 6 show various unripe forms in which the gradual casting off of the cytoplasm can be seen. (Compare with Fig. 4.) Cell 7 apparently was derived from a giant spermatid of which the large amount of cytoplasm still clings to the cell. Cells 8, 9 and 10 are abortive attempts at spermatozoal formation. Cell 10 shows vacuoles.

cytoplasmic masses of varying size (number 7 in Figure 5). They represent either a spermatid which has extruded its nucleus or cytoplasmic drops cast off by the developing spermatozoon. (Figure 6 shows these various stages of the casting off of the cytoplasm.) Such cytoplasmic masses are found not infrequently in small numbers in normal semen. They increase markedly with sexual overloading. In degenerative testicular processes they are very numerous in the early stages, but decrease later on. They again appear when regeneration sets in. Numbers 8, 9 and 10 are very abnormal and degenerating spermatids; 9, 10, 11 and 14 show multiple nuclei. Some also show degeneration vacuoles. Cell number 4 of the same figure approaches the shape of a sertolian cell, but I believe it to be a spermatid broken off a spermatoblast. The same is true of number 13 which shows various embedded sperms. Of course one must think of fused spermatids here also, that is, sperm agglutination in the sense of Stieve.[46] Another possible explanation is that active penetration of the spermatozoa (such as I have described in a lymphocyte[30]) may have taken place.*

semen. Furthermore, I have never seen any single testicular epithelial cell which I could definitively identify as a spermatocyte or spermatogonial cell.†

It seems to me that such cells could be loosened from their bed only in the severest types of testicular degeneration. In such an event, the cells themselves would be so degenerated that they would probably be absorbed before they completed their passage through the long tubular system of the male genital tract, or if really ejaculated would have become unidentifiable. Furthermore in testicular degeneration the spermatocytes are the first to show degeneration—cystic in type—as shown so beautifully by the work of Lagerloef.[42]

Interesting as the study of these testicular cells is, I want to stress here particularly their clinical significance when present in ejaculated semen. I have pointed out before that the difference between finding no

* After the present article was written I received two reprints from Dr. Ch. Joel (Michael and Joel: Zellformen in normalen und pathol. Ejakulaten und ihre klin. Bedeutung. *Schweiz. med. Wchnschr.*, 67: 757, 1937 and Pollak and Joel: Zur Morphol. d. maennl. Keimzellen. *Arch. f. exper. Zellforsch.*, 22: 77, 1938). These authors believe that these cell forms are evidence of spermatophagia which they think they have actually seen. I cannot argue this point, but I have never seen anything to suggest such spermatophagia and must consider the explanation given in the text as more probable.

† The authors cited in the previous footnote claim to have found spermatocytes and even spermatogonia in many abnormal semen ejaculates. This may be so, but their pictures of such cell forms do not seem convincing. It has been pointed out by many authors (Schinz and Slotopolsky,[49] Stieve,[46] and others) that the various cell forms of the testicular tubular epithelium are often very hard to differentiate. This is especially true when such cells occur in the ejaculated semen where they are nearly always degenerated.

sperms at all and only one or two is not just a few sperm cells. In the first place the finding even of one single sperm shows that at least some sperms are produced and in addition precludes occlusion of the excretory seminal ducts. Thus a puncture of the testicle to determine the presence or absence of spermatozoa in this organ becomes unnecessary. In the same way the presence of any of the testicular cell forms—forerunners of the spermatozoa—in the semen excludes occlusion of the seminal passages.

Testicular cells in the semen have a greater significance than that just mentioned. Given all the data of the case and the seminal picture, it is frequently possible to distinguish between certain forms of disturbed spermatogenesis and to evaluate the effect of a given treatment. A normal semen will show a very occasional spermatid and some protoplasmic drops, but I have not found more than one to five spermatids per 1000 spermatozoa counted in a normal specimen. The protoplasmic drops, while they are more frequently encountered than the spermatids, should not be abundant, since they occur mainly when unripe spermatozoa are ejaculated. Thus sexual overloading will lead to an increase of these protoplasmic drops. Generally the picture is completed by the presence of many unripened, coiled and improperly stained sperm heads. Such a picture might also be present in testicular hypoplasia, but the clinical data and rechecking of the seminal picture later on often make a differential diagnosis possible. Protoplasmic drops would naturally also be found in the early stages of acute testicular degeneration as Lagerloef[42] has shown in the bull. In addition one would also expect to find in such cases many long narrow and tapering and sickle shaped sperm heads and often almost a complete résumé of sperm pathology. A case from my practice may illustrate this:

Case III. H. N., male, white, 36 years old, came to me because, though he had been married eleven years, there were no offspring. Physical findings were negative, the testes being of average size and firm. The semen showed only about 50,000 sperms per c.c., and motility was only fair. The seminal morphology showed 50 per cent abnormal sperm heads with many narrow and tapering forms and an increased number of spermatids and protoplasmic drops, indicating as I believe, hypoplasia of the testicle and degeneration, at least in some areas.

Under treatment the patient improved. Three months later the amount of the semen ejaculate had definitely increased and the number of the sperms had risen to more than 1,000,000 per c.c. while the motility was much better. The abnormal sperm heads now totaled only a little over 30 per cent and there were fewer of the long tapering and narrow forms. The sickle-shaped heads had disappeared. Spermatids and protoplasmic drops had increased in number (evidence of regeneration).

Shortly after the last semen examination the patient contracted mumps with bilateral orchitis. Three weeks later the semen showed only about 10,000 sperms to the c.c. The motility was very poor. Most of the sperm heads were now abnormal with many very long narrow and tapering forms. Many protoplasmic drops and degenerated spermatids, often multinuclear, were found. The picture was that of an acute testicular degeneration.

As noted in parenthesis above, protoplasmic drops may also mean regeneration, either spontaneous or the result of treatment. In such cases where new sperms are being formed, many of them will at first be unripe and thus the number of the protoplasmic drops may indicate the effectiveness of the treatment, showing that active regeneration is taking place.

Case IV. F. P., white male, 30 years old, showed in the first semen specimen only 25,000 sperms to the c.c. with a few spermatids and protoplasmic drops. Under treatment the sperms increased to almost 3,000,000 to the c.c., while the number of protoplasmic drops about equalled the number of spermatozoa.

Spermatid giant cells and parts of the sertolian cell system always indicate degeneration in at least some of the tubules

of the testicle and the extent of such degeneration can be judged by the proportionate number of such cell forms seen. The presence of spermatocytes and spermatogonia would be a sign of further rapid and ultimate degeneration in the tubules of the gonad. Such a condition was present in Case III at the second examination made two months after the attack of orchitis. The semen was then only about one-half of the previous amount. Only one deformed immotile spermatozoon was found after a long search. Protoplasmic drops were practically absent, but degenerated spermatids, spermatid giant cells, many multinuclear and vacuolated, were seen. This is the only case in which I have seen spermatocytes and spermatogonia that I could identify definitely. The testicular tubule epithelium in some instances was cast off in sheets and the relative position of the various layers of the seminiferous tubule epithelium, though degenerated, could still be recognized.

Besides the seminal pictures described, there are also other semens which show few sperms with poor sperm morphology and practically no spermatids or protoplasmic drops. In such cases we are generally dealing with chronic testicular degeneration such as occurs in wasting diseases or is present in older men where spermatogenesis is definitely reduced.

In conclusion, I wish to emphasize that the evaluation of the sperm pictures as presented is, of course, only tentative and needs much more investigation before really definite answers can be given. Should it become possible to correlate various seminal findings and clinical lesions a most important advance would have been made from the diagnostic as well as the prognostic standpoint.

## SUMMARY

The number of sperms produced ordinarily is far in excess of that needed to fertilize the ovum.

Very long, tapering and narrow sperm heads are of especially sinister import and their shortening is often the first sign of improvement.

By evaluating the clinical data, the sperm morphology and the presence and number of the protoplasmic drops and seminiferous epithelial cells, it is frequently possible to distinguish between sexual fatigue, testicular hypoplasia and acute and chronic testicular tubule degeneration.

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# COMBINED INTRA-UTERINE, EXTRA-UTERINE PREGNANCY*

## WITH A REVIEW OF 291 CASES IN THE LITERATURE AND A REPORT OF THREE NEW CASES

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COMBINED pregnancy, i.e., simultaneous extra-uterine and intra-uterine pregnancy, is not so rare as many writers would lead one to believe. Parry,[25] in his monograph in 1876, stated that "twenty-two of the 500 cases of tubal pregnancy collected by him, were complicated by a coëxisting pregnancy."

The simultaneous occurrence of gestation within and without the uterus is, of course, a type of twin pregnancy, one fertilized ovum implanting itself in the uterus and one in the tube, or perhaps rarely in the ovary. The cause of this condition is some interference with the passage of the fertilized ovum from the ampulla of the tube, where fertilization usually occurs, into the uterus. Conception need not necessarily take place in the tube, for the spermatic particle may advance as far as the ovarian fimbriae to meet the ovum or, indeed, may penetrate the spot of rupture on the Graafian follicle and fertilize an ovum which has remained therein.[18–20] The spermatozoon may wander from the extremity of the tube on one side and fertilize an ovum from the opposite ovary, or an ovum of one ovary may be fertilized by a sperm cell in the opposite tube.[15]

The factors which may interfere with the passage of the fertilized ovum into the uterus are many. However, the most common cause from both clinical and pathologic evidence is some inflammatory lesion of the adnexa.

Many cases reported are not true combined pregnancy and have not been reviewed in this paper; we shall consider here only cases where the two pregnancies were concurrent and simultaneous.[16–17]

The first such case of combined pregnancy was reported by Duverney[13] in 1708, who discovered the condition at autopsy.

Since this report 290 further cases have been described. Strauss,[26] in 1898, collected thirty-two cases from the literature, while Weibel,[27] in 1905, increased the number to 119. In 1907, von Neugebauer[14] presented 169 cases, and in 1913[22] increased the number to 244. Novak,[6] in 1926, increased this number to 276 cases.

The present paper, while taking note of the articles published from 1926 to the present time, deals with only 294 cases: the 276 cases reported by Novak in 1926, together with fifteen cases since reported and three cases reported now.

The mortality was 20.7 per cent for the whole series, and 14.4 per cent if cases are excluded in which the condition was discovered only at post-mortem examination, and which did not receive treatment.

TABLE 1
AGE OF MOTHER

| Years | Cases |
|---|---|
| 20–25 | 29 |
| 26–30 | 57 |
| 31–35 | 70 |
| 36–40 | 40 |
| Over 41 | 8 |
| Unknown | 88 |

The maximum incidence is in the active period of childbirth, 26 to 35 years of age, with a tendency to fall in the latter half

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of the period. The previous parity (Table II) suggests that previous labors predispose to the condition.

TABLE II
PARITY OF MOTHERS

| | |
|---|---|
| Nullipara | 25 |
| Para I | 29 |
| Para II | 45 |
| Para III | 29 |
| Para IV | 12 |
| Para V | 10 |
| Para VI | 8 |
| Para VII | 4 |
| Para VIII | 1 |
| Para IX | 1 |
| Para X | 1 |
| Para XI | 1 |
| Para XII | 1 |
| Para XVIII | 1 |
| Multipara, figure not stated | 8 |
| Unknown | 116 |

The two pregnancies may occur at the same time or the uterine conception may antedate the extra-uterine, or vice versa. A normal pregnancy may occur while the dead products of an ectopic gestation are still present.

TABLE III
DURATION OF INTRA-UTERINE PREGNANCY

| Weeks | Cases |
|---|---|
| 4 | 2 |
| 6 | 16 |
| 8 | 28 |
| 10 | 10 |
| 12 | 36 |
| 14 | 9 |
| 16 | 7 |
| 18 | 2 |
| 20 | 6 |
| 22 | 4 |
| 24 | 7 |
| 26 | 3 |
| 28 | 7 |
| 30 | 1 |
| 32 | 3 |
| 34 | 2 |
| Term | 88 |
| Unknown | 60 |

The period of pregnancy varied from eleven weeks to term. In nine cases the cause of death was internal hemorrhage. Symptoms were present from a few hours in four cases to the whole duration of pregnancy in one case. All except one of these were recorded prior to 1897.

Forty-one cases were discovered after labor had started. In this group there were six deaths. In half of this group patients showed no symptoms except an unusual increase in the size of the abdomen. In six cases the intra-uterine pregnancy was terminated by abortion.

TABLE IV
DURATION OF EXTRA-UTERINE PREGNANCY

| Weeks | Cases |
|---|---|
| 2 | 0 |
| 4 | 1 |
| 6 | 14 |
| 8 | 56 |
| 10 | 3 |
| 12 | 33 |
| 14 | 1 |
| 16 | 4 |
| 18 | 2 |
| 20 | 1 |
| 22 | 0 |
| 24 | 0 |
| 26 | 0 |
| 28 | 2 |
| 30 | 1 |
| 32 | 0 |
| 34 | 0 |
| Term | 8 |
| Unknown | |

TABLE V

| | Patients Who Aborted, Weeks | Patients Whose Pregnancy Continued to Term, Weeks |
|---|---|---|
| Average duration of pregnancy at onset of symptoms | 8.3 | 7.2 |
| Average time of duration of symptoms before operation | 5.2 | 4.7 |
| Average time of preoperative bleeding | 3.5 | 1.2 |

TABLE VI
END RESULTS

| | No. of Cases | Recovered | Died |
|---|---|---|---|
| Mother | 226 | 182 | 44 |
| Intra-uterine fetus | 226 | 103 | 123 |
| Extra-uterine fetus | 215 | 7 | 208 |

In half of the patients after delivery a symptomless mass was noted in the abdomen. The other half had varying symptoms usually suggestive of infection

or intestinal irritation. In twelve cases the symptoms were so insignificant that nothing was done for the palpable mass. Eight patients passed the fetus piecemeal through fistulae, and of this group one died. A live child was removed from the abdomen on five occasions from one to twenty-three days after labor. Sixteen patients had exploratory laparotomies, two of whom died. In the other four patients who died, there had been no operative interference. The cause of death was peritonitis in three cases and intraperitoneal hemorrhage in the fourth.

Table VII
DIAGNOSIS

| When Made | No. of Cases | Deaths |
|---|---|---|
| Post-mortem | 16 | |
| Labor | 36 | 6 |
| Second half of pregnancy | 20 | 7 |
| First half of pregnancy | 219 | 16 |

In twenty patients the diagnosis was made in the second half of pregnancy or during labor. All were operated on and seven died. The causes of death were hemorrhage, septicemia, pulmonary embolism and pulmonary edema. Two deaths occurred in the sixth month, one in the seventh and four at term.

Table VIII
CASES DIAGNOSED IN FIRST HALF OF PREGNANCY

| | Cases | Total Mortality | Treatment | Mortality |
|---|---|---|---|---|
| After abortion of uterine ovum. | 47 | 7 | 39 laparotomies | 5 due to shock and hemorrhage |
| | | | 6 colpotomies | 0 |
| | | | 2 unoperated | 2 |
| Before abortion of uterine ovum. | 93 | 9 | 6 subtotal hysterectomies | 1 |
| | | | 3 curettage | 0 |
| | | | 83 salpingectomies | 8 |
| | | | 1 passed fetal parts by rectum | 0 |

## CASE REPORTS

Case I. A white multipara of 27 was admitted to Franklin Hospital July 26, 1933. About three months before she had had a three months miscarriage, and had last menstruated May 10, 1933. Three weeks before a small amount of vaginal bleeding had occurred, accompanied by nausea and vomiting, mild abdominal pain and low backache. A week later similar bleeding occurred with pain in the shoulders and neck. A diagnosis of left tubal pregnancy or threatened abortion was made. Spotting and abdominal pain over the left tubo-ovarian region persisted.

The pulse was 82 and regular, the blood pressure 114/70, the temperature 99°F. Abdominal examination showed moderate tenderness over the left tubo-ovarian region. No rigidity was present, but muscle guarding was noted. The cervix was soft with a slight amount of blood present. The uterus was one and one-half times normal size, firm, and in anterior position. The right fornix was clear, while the left showed a small tender mass.

At exploratory laporatomy under ether anesthesia free blood was found in the pelvis. The left tube was distorted in its middle third by a mass approximately 3 cm. in diameter. This was a pregnancy which had been bleeding through the fimbriated end. The left ovary was completely degenerated. The right tube showed areas of stenosis, resulting from an old pelvic inflammatory disease. Bilateral salpingectomy and left oöphorectomy were performed. The left tube was dilated into a sac which contained a two months fetus.

The patient made an uneventful recovery and was discharged in fourteen days. Her abdomen continued to enlarge and she did not menstruate. Six and-one half months later she was delivered of a normal child. (Case reported through courtesy of Drs. F. Foote and E. A. Gius.)

Case II. A multipara of 25 was seen May 15, 1936. She complained of irregular menstrual periods and was very desirous of becoming pregnant again. Her one child, aged 4, had been delivered by cesarean section.

She was fairly well developed and in no apparent pain. Abdominal examination was essentially negative. Pelvic examination showed an hypertrophied cervix and a small uterus in first degree retroversion. The fornices were negative.

The patient was given several tampon treatments and following her period on May 23, 1936 she was insufflated. The tubes were at first found closed, but after the third insuffla-

tion the tubes opened at 110 mm. of mercury. On September 14, she again appeared, stating that her last period had been June 29, and that she had missed the following two periods. Spotting had recurred on September 11, 1936.

Fig. 1. Preserved villi from tubal pregnancy. Case II.

The breasts were enlarged and colostrum could be expressed. The abdomen showed some tenderness to deep palpation in the left lower quadrant. The uterus was the size of a two months pregnancy and there was a small palpable mass in the left fornix, which I thought might be a cystic ovary.

The patient returned home, but spotting continued and the pain became aggravated. On September 17, she was doubled up with severe pain, bleeding intermittently. The abdomen showed muscle guarding and rigidity over the left lower quadrant. The uterus was still enlarged but a soft cystic and very tender mass was palpable in the left fornix. A diagnosis of left tubal pregnancy was made. The temperature was 99.2°F., the pulse 64, respirations 20, blood pressure 110/65.

Under gas and ether anesthesia the peritoneal cavity was opened and the old operative scar excised. The peritoneal cavity contained free fluid, but no blood. The right ovary and tube were negative. The uterus was enlarged to about a two months pregnancy and was in anterior position. The left tube in its distal third showed a bulging dark blackish mass, an unruptured eight week tubal pregnancy. The ovary on the left side was cystic. The cysts were punctured and left salpingectomy was performed. On section, the tube showed placental tissue and a small fetus. The peritoneal cavity was closed in the usual manner.

The patient made an uneventful recovery and was discharged from the hospital fourteen days postoperatively.

The intra-uterine pregnancy developed rapidly and normally, and on March 23, 1937 the patient was delivered of twin girls by cesarean section. At this time the remaining tube was ligated.

Thus the above findings show that three eggs must have been fertilized.

Case III. A multipara of 33, with one living child was seen by Dr. Lawrence Hoffman. She had had a miscarriage at about two and one-half months, and the flow was associated with some pain in the abdomen. This had become markedly aggravated.

A mass was found in the left cul-de-sac. An exploratory vaginal puncture was done and

blood was obtained. At laparotomy a left sided ruptured ectopic pregnancy was found and left salpingectomy was done. The patient made an uneventful recovery.

Both an extra-uterine and an intra-uterine pregnancy had been present.

## SUMMARY

1. Combined pregnancy occurs with reasonable frequency, as shown by the report of 294 cases.

2. The diagnosis at times is very difficult.

3. Surgery is the treatment of choice.

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# THE ACUTE ABDOMEN*

## WITH SPECIAL REFERENCE TO SYMPTOMS, DIAGNOSIS AND SURGICAL TREATMENT

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THE acute abdomen is one pathologic condition encountered in the general practice of medicine in which quick thinking and sound judgment are of paramount importance, as the mortality rate, in most instances, is in direct proportion to delay. In this paper the most important and most frequently encountered pathologic conditions will be discussed.

### ABDOMINAL INJURIES

Abdominal injuries, either open or closed, require immediate attention. Open injuries may be subdivided into (1) those which are limited only to the abdominal wall, and (2) those which enter the peritoneal cavity causing damage to the viscera. A stab or gunshot wound may enter the peritoneal cavity and cause no damage to the viscera but may introduce infection leading to a fatal peritonitis. For the same reason, probing of these wounds is dangerous.

The symptoms and signs of visceral injury are those of hemorrhage and shock, namely, pallor of the skin and mucous membranes, air hunger, quick, gasping respirations and sighing, cold skin with a clammy sweat, subnormal temperature, restlessness, dimness of vision, syncope and noises in the ears. The pulse is at first not affected but later becomes progressively more rapid, weak and of small volume. The blood picture will show a leucocytosis of from 19,000 to 30,000, with a diminution of the erythrocytes and low hemoglobin.

Babcock[1] differentiates shock from hemorrhage in the following manner: In shock there is a concentration of blood. There is a relative increase of hemoglobin and corpuscles, and an absence of leucocytosis. Air hunger is less marked, restlessness common and the patient more often unconscious. The patient in shock will usually show improvement when given the orthodox treatment in a reasonable length of time unless in extremis, while the patient suffering from hemorrhage will not, unless there is a spontaneous arrest or the bleeding is stopped by surgical intervention.

Closed abdominal wounds are diagnostic problems. A history of the nature of the accident is important. Symptoms of both shock and hemorrhage may be present. Physical examination reveals rigidity of the abdominal wall, hydro- or hemoperitoneum with distention and shifting dependent dulness. Pneumoperitoneum with diffuse tympany and obliteration of the liver dulness may be present. An empty leaking bladder may be found. A silent abdomen is usually diagnostic of peritonitis, whether due to blood, bile, urine or infection. The diagnosis is based upon history, symptoms and physical findings. *X*-ray examination is especially valuable in cases where there is a ruptured hollow viscus. The following is an illustrative case:

A young boy received a severe abdominal injury when the rear wheel of a heavily loaded truck passed over his abdomen. There was no visible external injury, but symptoms of hemorrhage were present, with painful, rigid abdomen, retraction of the knees and other signs of internal injury. Immediate laparotomy revealed about 5 inches of the mesentery torn from its attachment. The bleeding mesenteric vessels were ligated, the damaged bowel exteriorized, and the patient treated for hemorrhage and shock. Several weeks later the bowel was anastomosed. His life was saved by immediate, conservative surgery.

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The treatment of both open and closed abdominal wounds is surgical, when indicated. Open injuries involving only the abdominal wall may be treated conservatively. Meyer and Shapiro,[2] in a recent very instructive and extensive collective review, conclude in part as follows: "The policy of prompt exploration in penetrating wounds of the abdomen is established; however, no patient should be subjected to laparotomy until proper preparation has brought his systolic blood pressure above 80, better 90 or 100, unless the operation is one of 'last resort.' Secondary shock on a surgical service is equivalent to hemorrhage, and, until the bleeding vessels are ligated, should not be treated by intravenoclysis but by blood transfusion. Patients operated on in shock are less likely to survive the operative trauma. Correction of shock increases the resistance to peritonitis."

In gunshot or stab wound or in any injury to the bowel where there has been fecal contamination, the injection of 30 c.c. of coli-bactragen into the peritoneal cavity within three hours after the contamination will markedly diminish the chances of general peritonitis, and when bactragen is used it is not necessary to insert drains (Sternberg[3]).

Errors are too frequently made in attempting to do too much surgery in a patient suffering from hemorrhage or shock. Additional surgery can be performed in a second operation when the patient is a better surgical risk. It is much more logical to have a live patient with a fecal fistula or artificial anus than a dead one with a beautifully performed anastomosis.

## ACUTE APPENDICITIS

Acute appendicitis is one of the first acute abdominal conditions to be considered. In spite of the advancements made in surgical diagnosis and technique in the last few years, the mortality rate in this condition has increased. Abell[4] states that there are more than 20,000 deaths per year attributable to this disease, and Herrick[5] reports that in cases with peritonitis there is an operative mortality of 5 to 30 per cent.

As emphasized by Murphy, the symptoms of a typical attack of acute appendicitis, in chronological order, are pain, nausea, tenderness and fever. The pain is colicky in nature, reaching its height in about four hours; it may start in the epigastrium or may be diffuse over the abdomen, later becoming localized over McBurney's point. Nausea, which may be followed by vomiting, appears in from two to four hours after the onset of pain. Vomiting may occur only once or twice; however, if it is continuous there is probably present either a complication or another pathologic condition. The tenderness at first is diffuse and not marked, but in four to six hours it becomes definite, localizing over McBurney's point. Rebound tenderness usually indicates peritoneal inflammation, either local or generalized, and cutaneous hyperesthesia elicited by pinching a raised fold of skin over McBurney's point is occasionally present. The fever develops in from four to twenty-four hours after the onset of pain. Usually it is not high, ranging from 99 to 102 degrees, but more frequently remaining under 100 degrees. The more acute the infection, the earlier the temperature. A sudden drop in temperature suggests gangrene, perforation or evacuation of the appendiceal contents into the cecum.

Other symptoms should also be mentioned. Constipation is usually present, although Brunn[6] states that diarrhea is not uncommon in appendicitis. Diarrhea may be present if the exciting etiologic organism is pneumococcus or streptococcus, as well as in pelvic appendicitis and many times in appendicitis in children. Tachycardia is a very important clinical diagnostic point. A pulse rate of 90 to 120 is a more reliable evidence of infection than is increased temperature. Persistence of tachycardia with remission of other symptoms suggests gangrene. Bladder symptoms may result from contact inflammation, as when the

inflamed appendix lies in proximity to the bladder or ureter.

The findings on physical examination are right rectus muscular rigidity at McBurney's point, in direct proportion to the intensity and area of the underlying peritonitis. The position of the patient aids in the diagnosis; for instance, in acute septic appendicitis the patient lies on his back with the thighs elevated and the right knee or both knees flexed. This is an important and fairly constant guide in determining the severity of the attack. Pain on contraction of the right iliopsoas muscle by raising the right knee indicates a posterior or retrocecal appendix. Vaginal or rectal examination should be done. In children rectal examination is of particular value in establishing the diagnosis. Blood count will show a leucocytosis of from 9,000 to 18,000; this usually appears early with a relatively high proportion of polymorphonuclear leucocytes. Children under 4 years of age will usually show a relative lymphocytosis.

The diagnosis of acute appendicitis is made from the symptoms and the physical findings. In my opinion the clinical findings are more important in the diagnosis of acute appendicitis than are the laboratory findings. If the leucocyte count fits the clinical picture, use it; if not, use the clinical findings alone.

The treatment of acute appendicitis is surgery, and the earlier instituted the better. If delay has occurred and if the appendix appears to be sufficiently walled off by omentum, then postpone operation as the inflammatory mass may spontaneously resolve or a definite localized abscess may develop. If the latter occurs, drain the abscess but do not try to remove the appendix as the protective wall and the abscess may be broken and a generalized peritonitis develop. The appendix should be removed later when the infection has subsided. A ruptured appendix with generalized peritonitis presents a grave problem; operation with adequate drainage probably offers the patient the best chance of recovery. Miller[7] shows that in children with generalized peritonitis the mortality rate is lowered by operation and drainage.

In the treatment of ruptured appendix with peritonitis, I have obtained extremely gratifying results by the insertion of a catheter into the ileum, if possible, or into the cecum through the appendiceal stump. The catheter is held in place by a purse-string suture around the stump and brought out through the incision or through a stab wound. Distention is relieved by compressing the bowel, ileus is prevented, and the patient's chances for recovery are greatly improved. I have never had a fecal fistula develop following this method of treatment.

Decompression of the cecum as described above has been advocated by DeCourcy,[8] who shows a reduction in mortality to 4 per cent in a series of fifty cases.

Serum therapy has been used by Priestley and McCormack[9] in the treatment of generalized peritonitis secondary to rupture of the appendix with some hope of reducing the mortality from this complication.

## INTESTINAL OBSTRUCTION

Mechanical intestinal obstruction is the type of ileus which requires immediate surgery and, as in other acute abdominal conditions, the quicker the patient is submitted to surgery the better are the chances for recovery.

Intestinal obstruction may be caused by:

1. Pathology without the bowel, strangulated hernia either internal or external being the most common cause. Adhesive bands, either congenital or postoperative, are also frequently causative factors. Other causes are tumors (renal, splenic, pancreatic, ovarian and mesenteric), lymphatic inflammation, new growths, uterine and ectopic pregnancies, and occasionally gall- or urinary bladder stones.

2. Pathology within the bowel is caused from impacted feces, foreign bodies, parasites, hair balls, and gallstones which if

large enough will cause obstruction at the ileocecal junction.

3. Pathology developing from the wall of the bowel, such as tumor or carcinoma, is a common cause of intestinal obstruction. Carcinoma may be the cause of a chronic or partial obstruction and then may suddenly close the lumen, causing an acute obstruction. This is to be considered especially in patients within the cancer age group who give a history of abdominal distress with a change in bowel habit. The higher the obstruction the more severe will be the symptoms.

In children intussusception is the most common cause of obstruction. It is diagnosed by abdominal pain, early vomiting, palpable mass upon rectal examination with the characteristic "cranberry sauce" blood on the examining finger, associated with abdominal distention. Christopher[10] has recently reported two additional cases of intussusception in adults, one a high jejunal intussusception caused by a papillary adenoma and the other a chronic ileocecal intussusception.

Acute obstruction is a complication of regional enteritis, a recently described pathologic condition about which there is much discussion at the present time. Jackson[11] has reviewed the subject and reported several additional cases.

Other pathologic conditions, developing from the wall of the bowel and causing obstruction are strictures from healed ulcers, tuberculous and syphilitic lesions, chronic enteritis, diverticulitis and peridiverticulitis chiefly of the sigmoid and pelvic colons, benign tumors of the bowl, such as adenomata, lipomata, myomata, polyposis, endometrial adenomata and hemangioma. Mucocele of the appendix or hematoma of an appendix epiploica, while very rare, may give rise to acute obstruction.

The symptoms of intestinal obstruction are usually constant and include vomiting, projectile in type and containing first the contents of the stomach, then bile, and then intestinal contents. The loss of hydrochloric acid in the stomach contents leads to alkalosis, while the loss of pancreatic juice and bile leads to acidosis. The pain of the abdominal colic is centered around the umbilicus and is of a twisting, cutting, griping nature. It is constant with exacerbations if the obstruction is complete, while with incomplete obstruction there are remissions of the pain, and if gangrene is present there is temporary cessation of vomiting and pain. Meteorism or gaseous distention of the intestine is present except in high obstruction. No abdominal rigidity is present except when there is, an associated peritoneal irritation. A noisy abdomen with a metallic tinkling and splashing sound is characteristic in the early stages. Obstipation is a constant finding; the feces in the bowel beyond the obstruction may be passed but little or no gas will be expelled.

The diagnosis of acute mechanical obstruction is made by the presence of intermittent, crampy, colicky abdominal pain followed by nausea and projectile fecal vomiting, without tenderness, rigidity or fever. *X*-ray examination either by flat plate or by fluoroscopy may reveal step ladder-like arrangement of distended small bowel, especially if the obstruction is low. In acute cases, barium is not used in the *x*-ray examination.

The treatment of acute intestinal obstruction is surgical removal of the cause of obstruction as soon as possible after the diagnosis is made and with as little operative trauma and shock as possible. Prolonged and extensive operations are condemned. If a gangrenous piece of bowel is found, exteriorize it and do an enterostomy or colostomy. Later, when the patient is in better physical condition, the bowel can be anastomosed. When the viability of a piece of bowel is questionable, this piece may be brought up just beneath the skin surface in the incision and packed away from the abdominal cavity with gauze so as not to interfere in any way with the blood supply. If the viability of the bowel returns, nothing more need be done except to remove the

gauze and allow the wound to close. If, however, the questionable piece of bowel becomes necrotic, an enterostomy will spontaneously occur and, thereby aid in saving the life of the patient. Ileostomy, colostomy or cecostomy may be performed proximal to the obstruction; these procedures decompress the bowel and allow it to assume its normal size and function before any further surgery is attempted. Anastomosis should never be attempted in a piece of bowel which is markedly distended.

Sufficient fluids in the form of normal saline with or without glucose should be administered by venoclysis or hypodermoclysis to restore the lost chlorides. Preoperative administration is equally important with postoperative. The amount varies with the individual case and with the condition of the patient, but usually from 2,000 to 3,000 c.c. in twenty-four hours is sufficient. A Levine tube in the stomach with suction may be necessary to relieve the distention. Narcotics postoperatively may be used when necessary.

### PEPTIC ULCER

The term "peptic ulcer" includes both duodenal and gastric ulcer. A serious complication in this condition is hemorrhage which is usually treated medically. Allen[12] states that duodenal ulcers rarely cause fatal hemorrhage under the age of 50 years, while beyond this age the mortality is 33⅓ per cent. He advises the operative procedure of ligation of the artery supplying the ulcer in the better risk older group cases. Means[13] believes that operation should be limited to patients bleeding after 50 years of age or those having repeated hemorrhage.

Acute perforation occurs in a certain percentage of chronic ulcers. The symptoms are violent, acute abdominal pain which may cause the patient to fall to the floor or be unable to move. This pain may be referred to the epigastrium, right abdomen, scapula or back. Shock with pallid face, sweating brow, staring eyes may be present. The patient appears to be in agony and very apprehensive. Board-like abdominal rigidity is present, which is more marked over the ulcer or McBurney's point with the patient in a flexed position. These patients cannot lie flat; to avoid pain they lie in a flexed position. Breathing is shallow, jerky and costal. Temperature is normal or slightly subnormal. Pulse is slow, full and of normal volume until peritonitis develops. Vomiting is absent unless fluid or food is taken.

Physical examination reveals free fluid, rarely gas in the abdomen, obliteration of liver dulness, shifting dulness in the flanks and evidence of early peritonitis in the epigastrium and right iliac fossa. Vomiting of blood or its presence in the peritoneal cavity is unusual.

In acute perforation of an ulcer the diagnosis is made by the history of sudden and very violent epigastric pain, causing apparent shock, board-like rigidity of the muscles of the anterior abdominal wall, tenderness and dulness in the flanks and pelvis. Pneumoperitoneum may be present. There may be cessation or absence of abdominal peristalsis. The condition occurs most frequently in males who may or may not give an ulcer history. Flat *x*-ray plate or fluoroscopic examination usually shows air under the diaphragm and this is considered quite diagnostic.

The treatment of perforated peptic ulcer is immediate laparotomy to stop the leak. Any lapse of time after six hours from the time of perforation gives a poor prognosis, and after thirty-six hours the majority of patients die. Since these ulcers usually rupture on the anterior wall, the location of the perforation is quite evident, but because they generally are in shock and are poor surgical risks, the least amount of surgery done is good judgment, unless there are other indications. At operation the perforation is closed with a purse-string or superimposed Lembert suture. A piece of omental fat is attached over the sutured perforation and the abdomen is closed. A gastroenterostomy or any other

additional gastric surgery at this time is to be condemned, unless there is marked pyloric or duodenal obstruction. It is better judgment to perform any further gastric surgery, if necessary, at a later date when the patient is in better condition. The question of drains is controversial. Since the gastric contents are primarily sterile, many surgeons close the abdomen without drainage; others use drains and remove them early.

Postoperative fluids by venoclysis are essential. These patients should receive medical treatment for the ulcer following operation.

### RUPTURED ECTOPIC PREGNANCY

These patients present symptoms of early pregnancy and rupture usually occurs before the twelfth week. The prodromal symptoms are sharp, cutting, crampy, low abdominal pains progressing in severity as the pregnancy advances. Abnormal vaginal bleeding or spotting of blood is present in most cases. Decidual tissue may be passed, suggesting an incomplete abortion. Weak spells, sometimes severe enough to cause fainting, may be experienced. When rupture occurs there is sudden, severe abdominal pain, shock, collapse and usually evidence of intraperitoneal hemorrhage; the pulse rate is increased and of small volume; temperature is subnormal early but later may be elevated; vomiting sometimes occurs.

The Aschheim-Zondek or Friedman test will often determine the diagnosis before rupture or tubal abortion occurs. Bimanual examination reveals a soft and sometimes enlarged uterus. Marked tenderness in the pelvis over the affected adnexa and a palpable mass may be felt. The abdomen is tender and usually without rigidity. The cul-de-sac feels full and doughy. Posterior colpotomy with recovery of blood often aids in making a diagnosis. One can determine the presence of free blood in the peritoneal cavity by puncturing the posterior cul-de-sac with a large needle and aspirating its contents. Hope[14] has shown the value of the peritoneoscope in the differential diagnosis of this condition, and Ruddock[15] shows its value in the diagnosis of other abdominal and pelvic pathology.

Severe hemorrhage from a ruptured graafian follicle gives a similar clinical picture, except that the symptoms of pregnancy are lacking. The treatment, however, is usually the same as for ruptured ectopic pregnancy.

As soon as the diagnosis is established, immediate operation should be performed to stop the hemorrhage. Shock and depression may be out of proportion to the loss of blood and in those cases a delayed operation might be advisable until the patient is in better condition. This, however, is a point difficult to decide clinically. In most cases this condition should be considered as a surgical emergency. Evidence of intraperitoneal hemorrhage should indicate laparotomy. Removal of the ruptured tube and ligation of all bleeding points should be done as soon as possible, with immediate closure. Removal of the opposite tube or the appendix should not be done, except in the presence of gross pathologic changes when the condition of the patient permits further operation.

Blood transfusions are especially valuable in these cases and should be given after the bleeding is stopped. Shock is treated by venoclysis and hypodermoclysis along with the blood transfusions, and general supportive treatment should be instituted.

### ACUTE PELVIC CONDITIONS

Twisted ovarian cysts and uterine fibroids with impending gangrene are other pathologic pelvic conditions which require immediate surgery. Acute salpingitis is best treated conservatively.

### ACUTE CHOLECYSTITIS

The etiologic factors responsible for acute cholecystitis are (1) gallstones impacted in the cystic duct which interfere with emptying of the gall-bladder and at times produce, as a result of pressure,

interference with the circulation with resultant edema, congestion and occasionally gangrene and perforation; (2) hematogenous conditions, as from bacteria circulating in the blood stream during or following typhoid fever, septicemia, pneumonia and influenza. Rosenow's experiments led him to believe that streptococci in the blood stream have a peculiar elective affinity for the gall-bladder. (3) Bile infection from bacteria in the alimentary tract. Typhoid fever in earlier life has always been associated with this mode of infection. Potter[16] believes that biliary diseases in children may be caused by contributary factors, such as upper respiratory infections, including influenza, pneumonia, scarlet fever, appendicitis, intestinal parasites and sometimes a history of abdominal trauma. (4) Other theories suggested as a mode of infection are lymphatic-borne infections, infections due to propinquity from another organ, ascending biliary infections and amebiasis.

Acute cholecystitis is grouped pathologically by Taylor[17] into (1) acute edematous, (2) acute suppurative, and (3) acute gangrenous gall-bladder, which may be further subdivided into acute gangrenous infarct and acute suppurative gangrene. This grouping is based on the pathologic findings in the gall-bladder wall.

Symptoms of acute cholecystitis depend upon whether or not the cystic duct is obstructed by a stone. In the simple nonobstructive type the attack may be very slight and commonly is called acute indigestion or acute biliousness, rarely requires morphine for relief, and usually subsides under medical management. Acute obstructive cholecystitis is characterized by a sudden, severe attack of pain in the upper right abdominal quadrant or in the epigastrium and may radiate to the back in the region of the right eleventh rib, or to the right scapula. This pain is due to the passage of a stone down the cystic duct or its impaction in the neck of the gall-bladder. The pain is accentuated by the slightest diaphragmatic movement. Relief may be obtained by attempting a new position, as bending, putting pressure over the back, or by taking a hot bath. The intensity of the pain may become so great as to require morphine. Vomiting and constipation are almost always constant accompaniments. Vomiting may bring relief and is many times induced by the patient. Chills may be severe but the fever rarely rises above 102 degrees. The severity of the infection is indicated by the temperature. Marked rigidity over the right rectus muscle and extreme tenderness on pressure over the right upper quadrant are present. A palpable gall-bladder which moves with respiration may be found, but only if rigidity of the right rectus muscle is slight or absent. Jaundice is rare in uncomplicated cases.

The history of previous dyspepsia or distaste for certain foods, such as fats, with the above symptoms, is diagnostic of acute cholecystitis. The statistics of Mentzer[18] show that 72 per cent of the cases are in females. Leucocytosis of a moderate degree is usually present but many times is misleading as to the true underlying pathology and cannot be relied upon too much. *X*-ray by means of the gall-bladder dye or flat plate may be of value.

The treatment of acute obstructive cholecystitis is surgical, whether it be removal of the gall-bladder, drainage, or a combination of the two. In my opinion, all these methods have their merits in especially indicated cases and no set rule can be established. However, Puestow,[19] in recent experimental work, showed that removal of the gall-bladder causes a dilatation or paralysis of the sphincter of Oddi which allows free drainage from the bile passage, thereby reducing intraductal pressure. From this it appears logical that cholecystectomy should take precedence over the other methods. The present tendency is toward early cholecystectomy, as shown by the writings of Heyd,[20] Clute,[21,22] Mentzer,[18] Heuer,[23] and others. They believe that cystic duct obstruction is the first event in the pathologic process

with infection secondary to the obstruction. They therefore advocate removal before the secondary infection occurs.

Preoperative administration of glucose and saline should be given, preferably by venoclysis. This stores glycogen in the liver, corrects any abnormal chemistry in the body, and balances the body fluids. Postoperative care must include adequate fluid intake and the treatment of complications, if any occur.

### ACUTE PANCREATITIS

While this condition is rather infrequent, it is one of the most serious and carries one of the highest mortality rates of all acute abdominal conditions. There are several theories as to its etiology (Lewis[24]) but the "common channel theory" is the one most commonly accepted. Since it usually follows or is associated with cholelithiasis, it is thought that the ampulla of Vater becomes occluded or partially so, causing bile to flow from the common duct into the duct of Wirsung. The anatomic relations of these two ducts could in the majority of cases make this possible.

The pathology is a matter of the degree of destruction, ranging from acute hemorrhagic pancreatitis to acute gangrenous pancreatitis to acute suppurative pancreatitis, which may be diffuse or local.

The *onset* of symptoms is sudden and violent with agonizing, excruciating pain. The pain is so severe that large repeated doses of morphine may be required. Shock is a characteristic finding and in very severe cases is accompanied by cyanosis, pallor, cold and clammy skin. The temperature is subnormal with a rapid, weak pulse. Vomiting of bile and stomach contents usually occurs, and jaundice may appear early. The abdomen is distended and gives evidence of free fluid and early peritonitis. Epigastric tenderness and rigidity in the left upper quadrant is present.

Diagnosis is difficult, but the history of gall-bladder disease and sudden onset with shock are helpful along with an increase usually of the blood amylase.

DeKlimko[25] values the leucocyte count highly; it usually varies from 12,000 to 20,000. The symptoms closely simulate those of acute perforated peptic ulcer and acute cholecystitis and later bowel obstruction.

The treatment of acute pancreatitis is surgical. This consists usually of a right transrectus vertical incision. The purpose is to evacuate pancreatic fluid, clots, necrotic masses and to decrease the pressure of bile in the ampulla of Vater. Abell[26] states that the pancreatic tissue should not be interfered with but that gauze or drains should be inserted down to it. If the condition of the patient permits, the bile tract may be drained. This is especially indicated in the more chronic cases. Recently discussion has arisen as to the advantage of delayed operation. This is probably advisable in selected cases in the hands of selected surgeons, but due to the difficulty of correctly diagnosing the condition there probably would be many ruptured peptic ulcers diagnosed as acute pancreatitis by the average surgeon and hence delay would, in the majority of cases, mean the death of the patient.

### DIVERTICULITIS

Diverticulitis is inflammation of a preëxisting intestinal diverticulum. This diverticulum may be present in any part of the gastrointestinal tract and give no symptoms until complications arise. One frequently encounters Meckel's diverticulum located in the lower ileum about 30 to 150 cm. above the ileocecal valve. Occasionally it is lined, or at least partially so, by tissue which histologically is the same as gastric mucosa containing chief and acid cells secreting hydrochloric acid. Ulceration and perforation may occur in these cases.

Symptoms may be lacking, but when present, are due to complications, as catarrhal inflammation (Meckel's diverticulitis), ulcerations, hemorrhage, perforation or gangrene. In such instances the symptoms consist of pain, nausea, vomiting, localized tenderness, rigidity and in-

creased leucocyte count. These symptoms make it almost impossible to differentiate it from appendicitis. Other conditions which require differentiation are strangulation of the bowel as in a hernial sac, twists or attachments causing symptoms of bowel obstruction, and foreign bodies retained within the lumen.

The treatment for inflammation or obstruction is the operative removal of the diverticulum with closure as in appendicitis. Adherent or obstructed intestine should be freed or, if gangrenous, resected or exteriorized with an enteroanastomosis or enterostomy.

The colon, and especially the sigmoid, are the most frequent locations for herniations of the mucosa through weakened circular muscle. These herniations often enter the fatty tabs, cause rotation of the bowel toward the mesenteric attachment, collect and retain fecal masses, cause irritation, ulceration, and many times perforation. Carcinoma occasionally results.

Symptoms are chronic constipation, chronic catarrhal colitis with passage of blood-stained mucus. In acute inflammation there is sudden acute pain in the left lower quadrant or in the pelvis, with lower abdominal rigidity and sometimes a palpable tender mass, with fever and leucocytosis. Perforation may occur, usually into the bladder. *X*-ray and proctoscopic examination aid in the diagnosis, as does the history of an obese person over 50 years of age with chronic constipation.

Treatment is medical in about 85 per cent of cases, if there are no complications. Surgical treatment of the complications is often only palliative.

### MESENTERIC THROMBOSIS

Mesenteric thrombosis is fortunately an infrequent disease, attacking chiefly men past 40 years of age, usually with a history of myocarditis, endocarditis, or arteriosclerosis. The superior mesenteric artery is most frequently involved, rarely the inferior mesenteric, as shown by Gambee.[27]

Symptoms are sudden, violent, diffuse abdominal pain with collapse, followed by hematemesis and melena. Temperature is subnormal early but later rises with signs of ileus and peritonitis.

Diagnosis is extremely difficult and is rarely made preoperatively. If the patient has had previous heart disease or arteriosclerosis with the above symptoms, if there is a palpable mass, a leucocyte count of 20,000 or higher, with 85 per cent or more polymorphonuclear leukocytes, the diagnosis is more certain. Acute pancreatitis, perforated peptic ulcer and intestinal obstruction must be differentiated.

Treatment is immediate laparotomy, the necrotic bowel being exteriorized or resected. The entire intestinal tract should be carefully inspected so that no infarcts are overlooked.

### GENERAL CONDITIONS

The following are some conditions which may simulate an acute abdomen but do not require abdominal surgery: Renal colic, especially if on the right side, may closely simulate acute appendicitis; pneumonia and acute pericarditis are common causes of abdominal pain in children; food and lead poisoning may become confused with an acute surgical abdomen; tabetic crisis often simulates an acute abdomen in the adult; coronary disease, thrombosis, angina pectoris (Mohler,[28] Morrison,[29] Anderson,[30] Patterson,[31] Burns,[32] and Aynesworth[33]); spontaneous idiopathic hemopneumothorax (Hurxthal[34]); mesenteric lymphadenitis (Bell[35]); pulmonary tuberculosis (McReynolds[36]); juvenile diabetes mellitus (Newcomb[37]); blood dyscrasias, as Henoch's purpura, sickle-cell anemia and abdominal allergy (Althausen, Deamer and Kerr,[38] Campbell,[39] Davis[40]); subarachnoid hemorrhage (Thurston[41]); hemorrhage into a hydatid of Morgagni (Zener[42]); arachnoidism (Morton[43]) and periarteritis nodosum (Sawyer[44]).

### SUMMARY

1. Pain is the chief symptom of the acute abdomen.

2. Early diagnosis of an acute surgical abdomen is essential.

3. The most frequent and important surgical conditions of the abdomen have been discussed.

4. Diseases both common and rare which give abdominal pain but do not require abdominal surgery have been mentioned.

5. Immediate conservative surgery, when indicated, has been stressed.

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# PEPTIC ULCER OF MECKEL'S DIVERTICULUM

## CASE REPORT AND REVIEW OF THE LITERATURE

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ALTHOUGH peptic ulcer of Meckel's diverticulum, giving rise to its various late symptoms, has been known to the pathologist for almost a century, it has been only during the last decade that clinicians have attempted to develop a definite symptom-complex that would enable them to diagnose preoperatively a condition which, judging from published reports of the past few years, is by no means as rare as the textbooks would have us believe.

Uncomplicated Meckel's diverticulum has been described as occurring in 1.5 to 3 per cent of all persons, being present, according to Harbin,[27] from 14 to 16 inches (35 to 40 cm.) from the cecum. Normally the vitelline duct undergoes complete obliteration during the seventh week, but the duct, or segments of it, may persist. When the remnant of the primitive duct proximal to the ileum persists, it forms what is called Meckel's diverticulum. Heterotopic tissue is present in approximately 25 per cent of all Meckel's diverticuli.

Numerous theories have been proposed to explain the presence of such aberrant elements as gastric and duodenal mucosa and even pancreatic tissue "rests." The earliest and most commonly accepted theory is that of Albrecht[3] which maintains that the entoderm lining the primitive intestinal tube possesses the potentiality of developing into any of the glandular components of the fully developed gastrointestinal tract. Shaetz[56] proposes that during early embryonic life there may be a transference of endodermal cells lining the primitive intestinal tube, due to the rotating movements of the embryo, with reimplantation of these cells at narrowed points in the intestinal tract. Farr and Penke[18] hold that the vitello-intestinal duct originally may have had a digestive function and hence embodied a complete primitive digestive system.

Greenblatt, Pund and Chaney[23] add the theory of "dysembryoma" in which they agree with Albrecht that the entodermal lining does form cell groups which function as a distinct primitive digestive system such as Farr and Penke suggest. They go one step farther in stating that while normally this embryonic system retrogresses as soon as its function ceases, occasionally "a vestige of heterotopic tissue remains as a consequence of retarded embryological retrogression of the omphalomesenteric duct." Certainly the nutritive elements within the early yolk sac are assimilated by the embryo and we are forced to agree that these elements certainly must be acted upon by the cells of the vitelline duct in some manner analogous to passage of food through the upper digestive tract before it is absorbed by the cells of the jejunum and ileum.

When present, the heterotopic tissue usually lines the greater portion of the diverticulum, often involving the neck of the sac. Hence surgical removal should not consist of merely excising the anomaly. A small length of ileum extending to each side of the diverticulum should be removed, and anastomosis, preferably lateral, performed; otherwise we cannot be sure that all of the gastric or duodenal cells, if present, have been removed. The patient then runs the risk of later developing a peptic ulcer of the ileum with a repetition of his original symptoms.

Greenblatt and his associates emphasize the difficulty of correct diagnosis because of the possible various symptom combinations which may so closely imitate one of several different and more common acute abdominal conditions. They have analyzed a series of eighteen cases of Meckel's diverticulum observed during the past ten years at the University Hospital of the University of Georgia School of Medicine. They divide these into six groups. (Table I.)

Meckel's diverticulum with peptic ulcer, *can* be made, as we will attempt to show. The cardinal symptoms and signs are: (1) history of massive intestinal hemorrhage; (2) an average age of from 10 to 15 years; and (3) abdominal pain.

Practically all writers agree that the cardinal symptom of peptic ulcer of a Meckel's diverticulum is hemorrhage. It was present in twenty-nine of the thirty-four cases reported by Greenwald and

TABLE I

| Group | Findings | Symptomatology |
|---|---|---|
| Peptic | Gastric mucosa: without ulceration; with ulceration; ulcer and hemorrhage; ulcer and perforation | May simulate duodenal ulcer. History of intestinal hemorrhage. Peritonitis due to perforation. |
| Obstructive | Intussusception<br>Volvulus<br>Bands and adhesions<br>Contents of inguinal or femoral hernia | Signs and symptoms of intestinal obstruction may vary from chronic to acute; partial to complete obstruction. |
| Diverticular | Simple acute<br>Acute with perforation and gangrene<br>Chronic | Symptomatology essentially that of appendicitis. |
| Umbilical | Fecal fistula<br>Umbilical adenoma<br>Prolapse of intestine through umbilical fistula | Lesions of umbilicus often associated with underlying omphalomesenteric duct. |
| Tumor | Benign: enterocystoma; carcinoid; adenoma; mesodermal tumors<br>Malignant: carcinoma; sarcoma<br>Heterotopic: pancreatic tissue; embryonal rests | Symptoms of bleeding, intussusception or obstruction. |
| Incidental | Normal intestinal structure | None |

Clinically the diagnosis of groups two, three, five and, naturally, six is practically impossible because of the absence of any clear cut differential sign that might rule out the more frequently encountered abdominal lesions. The diagnosis of group four becomes possible because the presence of tumors, fistulas or cysts of the umbilicus would suggest the presence of a patent omphalomesenteric duct within the abdomen.

The diagnosis of the lesions in group one, Steiner.[24] In five of these cases it was the only symptom and extended over a period of from one day to nine months. In a second group of five cases the initial symptom was intestinal hemorrhage which was subsequently followed by abdominal pain. The hemorrhages were of longer duration in this group and were present for from two months to four years. In the third group of five cases the onset occurred with abdominal pain but with hemorrhages appearing

later. In this group the course was more acute, the duration of the bleeding being given as from twenty-four hours to two weeks. Of the group of seventy-seven histologically proved cases in Chart I sixteen, or approximately 20 per cent, contain no mention of intestinal hemorrhage having been present. An analysis of these cases (Table II) reveals the interesting

TABLE II
ANALYSIS OF CASES IN WHICH NO HEMORRHAGE WAS REPORTED

| Case No. | Preoperative Diagnosis | Gross Findings |
|---|---|---|
| 1 | Perforated appendix | Perforated ulcer at base; peritonitis. |
| 4 | Appendicitis | Perforated ulcer at edge of gastric mucosa. |
| 15 | Appendicitis; peritonitis | Perforated ulcer partly walled off. Local peritonitis |
| 21 | Perforated appendix | Perforated ulcer at base with peritonitis. |
| 23 | ? | Umbilical cyst containing gastric juice. Ulcer at base. |
| 24 | Peritonitis | Perforated peptic ulcer. Peritonitis. |
| 26 | Peritonitis | Two ulcers at neck; one perforated. |
| 29 | Perforated appendix | Perforated ulcer at base with peritonitis. |
| 35 | Peritonitis | Perforated ulcer at base found at autopsy; peritonitis. |
| 37 | ? | Chronic ulcer at neck of diverticulum. |
| 40 | Acute appendicitis | Perforated ulcer. Walled off. |
| 42 | Peritonitis, appendiceal | Adherent mass. Lentil shape base sealed to abdominal wall. |
| 43 | ? | Chronic peptic ulcer at base of diverticulum. |
| 47 | Acute appendicitis | Lentil shape perforation at base sealed to abdominal wall. |
| 58 | Duodenal ulcer with adhesions | Superficial erosion of mucosa of diverticulum. |
| 71 | Acute appendicitis | Perforated ulcer of base of diverticulum. |

observation that in all but three cases the ulcers *had already perforated*, causing either local or diffuse peritonitis. Of the three remaining unperforated cases, two (Case 23 and Case 58) would, perhaps, not belong to the peptic group of Meckel's diverticulum according to Greenblatt's classification. It would be interesting to review the history of each of these cases now that the emergency of an acute abdomen has passed; perhaps the examiner's questioning, no longer prejudiced by the patient's appearance, temperature and rigid abdomen, would have taken a wider view and elicited the fact that hemorrhage *had* been present at some time or another prior to hospitalization of the patient.

Usually the patient gives a history of previous massive intestinal hemorrhages followed by a relatively long period—months and even years—before recurrence. The hemorrhage is usually massive although in some cases only slightly bloody stools have been seen. The stool is composed of bright red to black blood and may be fluid or semi-clotted. It is not mixed with mucus as in the dysenteries, nor does it have the raspberry jam appearance of intussusception. Alarmingly rapid exsanguination to the point of collapse indicates the seriousness of the condition. Moll[50] reported a case in which the bleeding began two days after birth and continued intermittently up to five months when laparotomy was performed. Callender[10] reported the case of an infant who died thirty-six hours after the first hemorrhage. However, hemorrhage may persist over a long period of time. Megevaud and Dunant[47] cite cases in which hemorrhage had occurred intermittently from childhood to adult life.

Conditions besides *intussusception* and the *dysenteries* which may simulate bleeding peptic ulcer of a Meckel's diverticulum are: *Henoch's purpura* which may be ruled out by the absence of purpuric spots, normal bleeding and clotting times, normal clot retraction and normal platelet count; *bleeding peptic ulcer* is somewhat more difficult to eliminate because some cases of diverticular ulcer do have a definite pain-food relation. However, the pain of duodenal ulcer is usually epigastric in position, there is a more prompt response to the

effects of the alkalis, and the duodenal ulcer will usually be revealed by *x*-ray study. *Rectal polyp* may bleed profusely but a history of tenesmus and the aid of the proctoscope may prove the absence of this lesion. The *x*-ray may or may not be of value in outlining the mass. *Ruptured rectal varicosities* may cause sudden massive hemorrhage similar to the bleeding diverticular ulcer, but here again the proctoscope affords a means of differentiation. *Congenital intestinal telangiectasis* is so rare that it is mentioned only to be dismissed as mathematically improbable. It is our opinion that, regardless of other symptoms, a history of massive hemorrhage from the rectum followed by recovery and then recurrence of the hemorrhage after the lapse of weeks or months is almost pathognomonic of peptic ulcer of a Meckel's diverticulum.

Table III
Age Incidence of Peptic Ulcer of Meckel's Diverticulum

| Age | No. of Cases | Percentage |
|---|---|---|
| 1 or less | 8 | 10.8 |
| 1 to 10 | 34 | 45.9 |
| 11 to 20 | 21 | 28.3 |
| 21 to 30 | 9 | 12.1 |
| 31 to 40 | 0 | 0 |
| 41 to 50 | 2 | 2.7 |
| Age not reported | 2 | 2.7 |

Peptic ulcer of a Meckel's diverticulum is an affection of childhood. The first and second decades of life are most frequently involved although no age group is immune. Kleinschmidt[37] reported a case in a patient 45 years old, and McKeen had a patient 53 years old in whom this condition was associated with duodenal pathology. In the series of cases summarized in this paper approximately 75 per cent were found in patients less than 20 years old, with the greatest number of cases occurring in the age group of from 1 to 10 years. (Table III.)

Johnston and Renner's[36] excellent review, which we have taken the liberty to use as the basis of our compilation of cases, shows that the ulcers were discovered principally in infants and children: 14 per cent were less than 1 year old; 18 per cent were between 1 and 2 years; 5 per cent between 3 and 4; 23 per cent were 5 to 10 years of age; 24 per cent from 11 to 16; and the remainder from 16 to 53 years of age. The age was not given in two cases, although Treplin's case is reported as being a child.

Pain is present in a majority of the cases but may vary from mild abdominal distress on to the agonizing paroxysms of peritonitis. Of course the extreme degree of abdominal pain only follows perforation of the anomalous viscus. This symptom would be rendered obsolete if the surgeon recognized the underlying pathologic condition in its unruptured stage. The most frequent type of pain encountered is a colicky cramp-like sensation referred to the umbilical or hypogastric regions, and is probably due to distention of the small bowel or colon by the enormous amount of blood and clots within their lumen; however, several cases reported indicate a definite relationship of pain to food, just as in duodenal ulcer. Kleinschmidt's patient developed pain regularly one and a half hours after meals; Megevaud and Dunant's patient was relieved by food, and Greenblatt et al. report in their series the case of a man 47 years old whose pain was regularly relieved by doses of sal hepatica. The pain, regardless of degree, may precede or follow the hemorrhage and usually bears no relationship to meals.

Tenderness and rigidity are absent and palpation may either cause a moderate amount of discomfort or may be negative. Perforation of the ulcer, of course, will yield a picture of peritonitis and an etiologic diagnosis, except possibly from the history, becomes impossible. Pain, therefore, while a valuable aid to the diagnosis of peptic ulcer of Meckel's diverticulum, if it occurs in the classical manner, may manifest itself in so many possible degrees and characterizations that

# CHART I

## REVIEW OF HISTOLOGICALLY PROVED CASES

| Author | Year | Age | Sex | Symptoms | | | Preoperative Diagnosis | Gross Findings | Histologic Findings | Result | Remarks |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | Pain | Intestinal Hemorrhage | Acute Abdomen | | | | | |
| 1. Deetz[15] | 1907 | 9 | M | + | − | + | Perforated appendix. | Perforated ulcer at base. Peritonitis. | Gastric and pancreatic mucosa. | Recovered | First definite clinical case. |
| 2. Huebschmann[31] | 1913 | 4½ | M | + | + | + | Peritonitis. | Perforated marginal ulcer. Peritonitis. | Ulcer at edge of gastric mucosa. | Died | |
| 3. Callender[10] | 1915 | 19 mo. | .. | − | + | − | Autopsy. Death of hemorhage. | Punched out ulcer at base; eroded vessel. | Gastric and ileac mucosa. | Died | |
| 4. Gramen[22] | 1915 | 10 | M | + | − | + | Appendicitis. | Perforated ulcer. Peritonitis. | Ulcer at edge of gastric mucosa. | Recovered | |
| 5. Meulengrecht[49] | 1918 | 12 | M | + | + | − | Autopsy. Death of other cause. | Ulcer about to perforate. | Ulcer at edge of gastric mucosa. | Died | |
| 6. Mueller[51] | 1919 | 11 | M | + | + | + | Peritonitis. | Perforated ulcer. | Ulcer at edge of gastric mucosa. | Recovered | |
| 7. Megevaud and Dunant[47] | 1922 | 28 | M | + | + | − | Duodenal or intestinal ulcer. | Ulcer at base of diverticulum. | Ulcer at junction of gastric and ileac mucosa. | Recovered | |
| 8. Schreuder[55] | 1923 | 8 | M | + | + | − | Duodenal ulcer (x-ray). | Thickened pylorus. Ulcer at base of diverticulum. | Pancreatic tissue at tip. | Recovered | |
| 9. Brasser[5] | 1924 | 15 | M | + | + | + | Tumor or polyp. Autopsy. | Perforated ulcer at base. Peritonitis. | Ulcer at junction of gastric and ileac mucosa. | Died | Perforated 8 days after exploration for tumor or polyp. |
| 10. Guibal[25] | 1924 | 14 | M | − | + | − | Intestinal tuberculosis. | Chronic penetrating marginal ulcer. Eroded vessel. | Ulcer at junction of gastric and ileac mucosa. | Recovered | |
| 11. Humbert[34] | 1924 | 11 mo | M | + | + | + | Peritonitis. | Perforated ulcer. Peritonitis. | Ulcer at junction of gastric and ileac mucosa. | Died | |
| 12. Pascale[52] | 1925 | 41 | M | + | + | − | Ulcus simplex. | Ulcer at Merckel's diverticulum. | Peptic element confirmed. | Recovered | |
| 13. Ulrich[62] | 1925 | 8 mo. | M | − | + | − | Peritonitis. | Perforated ulcer. Peritonitis. Blood in peritoneal cavity. | Bleeding ulcer at base; gastric mucosa present. | Died | Perforated while under observation. |
| 14. Abt and Strauss[1] Case number 1. | 1926 | 2 | F | + | + | − | ? | Ulcerated and inflamed diverticulum. | Gastric mucosa at apex; infiltrated and fibrotic as around peptic ulcer. | Recovered | |
| 15. Etchegorry[17] | 1926 | 16 | M | + | − | + | Appendicitis; peritonitis. | Perforated ulcer partly walled off. | Gastric mucosa distal to ulcer of diverticulum. | Recovered | |
| 16. Kleinschmidt[37] | 1927 | 15 | M | + | + | + | Appendicitis. | Perforated ulcer at base. Peritonitis. | Gastric mucosa present. | Recovered | Perforated while under observation. |
| 17. McCalla[40] | 1927 | 4 | M | + | + | + | Autopsy. | Perforation at base. Abscessed abdominal wall. Peritonitis. | Ulcer at junction of gastric and ileac mucosa. | Died | Perforated while under observation. |
| 18. Jackson, A. S.[35] | 1927 | 14 | M | + | + | + | Ulcer of Meckel's diverticulum. | No gross ulcer. | Gastric mucosa and area of ulceration. | Recovered | First case diagnosed clinically. |
| 19. Taylor[58] | 1927 | 17 mo. | M | − | + | − | Autopsy. | Intramesenteric tumor with overlying chronic ileac ulcer. | Ulcer bordering a zone of gastric mucosa. | Died | Exsanguination. |
| 20. Meiss[48] | 1928 | 2 | M | − | + | − | Intussusception or polyp. | Chronic ulcer at tip perforating ileum at a higher level. Ileum full of blood. | Gastric and ileac mucosa. | Recovered | |
| 21. Hartglass[28] | 1928 | 14 | F | + | − | + | Perforated appendix. | Perforated ulcer at base. Peritonitis. | Ulcer at junction of gastric and ileac mucosa. | Recovered | |
| 22. Peterman and Seeger[53] | 1928 | 6 | M | + | + | + | Ulcer of Meckel's diverticulum. | Perforated chronic marginal ulcer; ulcer at tip perforating ileum at higher level, also two ileac ulcers. | Gastric mucosa present. | Recovered | At first operation two weeks previously blood in peritoneal cavity. |
| 23. Treplin[61] | 1929 | child | ? | − | − | + | Previous appendectomy; second operation for recurrent pain. | Umbilical cyst containing gastric juice. Peptic ulcer at neck of diverticulum. | Ulcer in ileac mucosa; islets of gastric mucosa. | Recovered | |

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 24. Franke[20] | 1929 | ? | ? | − | − | + | Peritonitis. | Perforated ulcer. Peritonitis. | Ulcer in ileac mucosa. Cap of diverticulum lined with gastric mucosa. | Died | |
| 25. Winkelbauer[64] | 1929 | 2 | M | + | + | − | Intussusception. | Indurated Meckel's diverticulum. | Shallow ulcer near island of gastric mucosa. | Recovered | |
| 26. Schmidt[54] | 1930 | 18 | M | − | − | + | Peritonitis. | Two chronic ulcers at neck, one perforated. | Ulcers at junction of gastric and ileac mucosa. | Recovered | |
| 27. Aschsner and Karelitz[2] | 1930 | 15 mo. | F | − | + | − | Ulcer of Meckel's diverticulum. | Penetrating marginal ulcer. Mesenteric adenitis. | Ulcer in ileac mucosa at edge of gastric mucosa. | Recovered | |
| 28. Achsner and Karelitz[2] | 1930 | 2 | M | − | + | − | Intussusception. | Peptic ulcer at base. | Ulcer at junction of gastric and ileac mucosa. | Recovered | |
| 29. Fevre, Patel and Lipart[19] | 1930 | 7 | M | + | − | + | Perforated appendix. | Perforated ulcer at base. Peritonitis. | Chronic ulcer; duodenal ulcer present. | Recovered | |
| 30. Fevre, Patel and Lipart[19] | 1930 | 5 mo. | M | + | + | + | Intussusception. | Perforated ulcer at base. Peritonitis. | Ulcer at junction of gastric and ileac mucosa. | Recovered | |
| 31. Haberer[26] | 1930 | 13 | M | + | + | + | Postoperative appendiceal abscess. | Perforated ulcer with local abscess. | Ulcer at junction of gastric and ileac mucosa. | Recovered | Operated upon one month previously for appendicitis. |
| 32. Haberer[26] | 1930 | 21 | M | − | + | − | Bleeding duodenal ulcer. | Two duodenal ulcers; bleeding diverticular ulcer. | Gastric mucosa present. | Recovered | |
| 33. Cobb[12] | 1931 | 18 | M | + | + | + | Acute appendicitis. | Punched-out, perforated marginal ulcer walled off. | Ulcer at margin of gastric and ileac mucosa. | Recovered | |
| 34. Tavernier and Guilleminet[57] | 1931 | 10 | F | − | + | − | Possible ulcer of Meckel's diverticulum. | Small ulcer at base. | Gastric mucosa distal to ulcer. | Recovered | Preoperative *x*-ray series negative. |
| 35. Lindau and Wulff[39] | 1931 | 15 | M | + | − | + | Peritonitis. | Peritonitis. Perforated ulcer found at autopsy. | Chronic perforating ulcer adjacent to gastric mucosa. | Died | |
| 36. Greenwald and Steiner[24] | 1931 | 15 wk. | M | + | + | + | Perforated ulcer of Meckel's diverticulum. | Punched-out perforation near base. | Areas of gastric mucosa. | Died | |
| 37. Beuchner[8] | 1931 | 8 | F | + | − | − | ? | Chronic ulcer at neck of diverticulum. | Gastric mucosa present. | Recovered | |
| 38. Hudson and Koplik Case 12[32] | 1932 | 6 | M | + | + | + | Peritonitis. | Perforated ulcer. Peritonitis. | Ulcer at junction of gastric and ileac mucosa. | Died | Perforated while under observation. |
| 39. Hudson and Koplik Case 31[32] | 1932 | 5½ | M | + | + | − | ? | Sigmoid full of blood. Inflamed diverticulum. | Inflammation and superficial ulceration; gastric and ileac mucosa present. | Recovered | |
| 40. Vaughn and Singer[63] | 1932 | 7 | M | + | − | + | Acute appendicitis. | Walled-off perforation. | Ulcer at junction of gastric and ileac mucosa. | Recovered | |
| 41. Mason and Graham[45] | 1932 | 9 mo. | M | − | + | − | ? | Meckel's diverticulum. | Chronic ulcer at junction of gastric and ileac mucosa. Pancreatic cells. | Recovered | |
| 42. Houdil and Marty[30] | 1932 | 7 | M | + | − | + | Peritonitis, appendiceal. | Adherent mass. Lentil-shaped perforation at base. | Gastric mucosa present. | Died | |
| 43. Wulff[65] | 1932 | 16 | F | ? | ? | ? | ? | Chronic peptic ulcer at base. | Gastric mucosa present. | Recovered | |
| 44. Hudson[33] Case 1 | 1933 | 11 | M | + | + | − | ? | Blood filled diverticulum. | Ulcer at junction of gastric and ileac mucosa. | Died | |
| 45. Hudson[33] Case 3 | 1933 | 6 mo. | M | + | + | + | Peritonitis. | Perforation of diverticulum. Peritonitis. | Gastric mucosa present. | Died | |
| 46. Dragstedt[16] | 1933 | 17 | M | + | + | − | Ulcer of small intestine. | Chronic perforating ulcer. | Diverticulum lined with gastric mucosa. | Recovered | |
| 47. Johnston and Renner[36] | 1933 | 18 mo. | M | + | − | + | Acute appendicitis. | Acute lentil-shaped perforation at base sealed against abdominal wall. | Ulcer at junction of gastric and ileac mucosa. | Recovered | |
| 48. Johnston and Renner[36] | 1933 | 15 mo. | F | − | + | − | Ulcer of Meckel's diverticulum. | Superficial ulcer at base. | Superficial shallow ulcer at junction of gastric and ileac mucosa. | Recovered | |
| 49. Chesterman[11] | 1935 | 22 | M | − | + | − | Exploratory laparotomy. | Meckel's diverticulum and tuberculous glands. | Ulcer at junction of gastric and ileac mucosa. | Recovered | |
| 50. Chesterman[11] | 1935 | 11 | F | + | + | − | Peptic ulcer of Meckel's diverticulum. | Peptic ulcer of Meckel's diverticulum. | Gastric mucosa. | Recovered | |
| 51. Chesterman[11] | 1935 | 6 | F | − | + | − | Possible peptic ulcer of Meckel's diverticulum. | Peptic ulcer of Meckel's diverticulum. | Gastric mucosa. | Recovered | |

## CHART (*Continued*)

### REVIEW OF HISTOLOGICALLY PROVED CASES

| Author | Year | Age | Sex | Symptoms | | | Preoperative Diagnosis | Gross Findings | Histologic Findings | Result | Remarks |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | Pain | Intestinal Hemorrhage | Acute Abdomen | | | | | |
| 52. Chesterman[11] | 1935 | 16 | M | + | + | + | Peptic ulcer of Meckel's diverticulum. | Peptic ulcer of Meckel's diverticulum. | Gastric mucosa. | Recovered | |
| 53. Cobb[13] | 1936 | 10 | M | + | + | + | Perforated ulcer of Meckel's diverticulum. Appendicitis. | Acute appendicitis and Meckel's diverticulum. | Gastric mucosa with small peptic ulcer. | Recovered | |
| 54. Cobb[13] | 1936 | 1½ | M | + | + | + | Ruptured viscus. | Perforated Meckel's diverticulum. Peritonitis. | Gastric mucosa with small peptic ulcer. | Died | |
| 55. Brown and Pemberton[7] | 1936 | 6 | M | − | +(?) | − | Meckel's diverticulum (?). | Meckel's diverticulum. | Diverticulitis with gastric mucosa. | Recovered | Previous appendectomy. |
| 56. Brown and Pemberton[7] | 1936 | 7 | F | + | +(?) | − | Meckel's diverticulum or polyp. | ? | Gastric mucosa with inflammation. | Recovered | |
| 57. Brown and Pemberton[7] | 1936 | 9 | F | + | +(?) | − | Lesion of small bowel. | ..... | Gastric mucosa with inflammation. | Recovered | |
| 58. Brown and Pemberton[7] | 1936 | 15 | M | + | + | .. | Lesion of small bowel. | ..... | Gastric mucosa but no acid cells. | Recovered | Previous appendectomy. |
| 59. Brown and Pemberton[7] | 1936 | 22 | M | + | + | .. | Meckel's diverticulum. | ..... | Ulcer at base of diverticulum containing gastric mucosa. | Recovered | |
| 60. Mallory[43] | 1936 | 10 mo. | F | − | + | − | Ulcer of Meckel's diverticulum. | Meckel's diverticulum. | Gastric mucosa present. | Recovered | |
| 61. Mallory[44] | 1936 | 22 | M | + | + | − | Meckel's diverticulum. | Ulcer at base of Meckel's diverticulum. | No gastric mucosa seen. | Recovered | |
| 62. Greenblatt, Pund, and Chaney.[23] | 1936 | 47 | M | + | − | − | Duodenal ulcer with adhesions. | Diverticulum with superficial erosion. | Gastric mucosa and pancreatic tissue. | | |
| 63. Greenblatt, Pund, and Chaney.[23] | 1936 | 23 | F | + | + | − | Diverticulitis; postoperative adhesions. | Meckel's diverticulum. | Gastric mucosa. | Recovered | |
| 64. Greenblatt, Pund, and Chaney.[23] | 1936 | 28 | F | + | + | + | Intestinal obstruction. | Intussusception of ileum; Meckel's diverticulum with ulcer. | Gastric mucosa. | | |
| 65. Breccia[8] | 1936 | 23 | M | + | + | + | Acute appendicitis. | Meckel's diverticulum with ulcer. | Gastric mucosa and duodenal glands. | Recovered | |
| 66. Bagwell (Curd)[4] | 1936 | ? | M | ? | ? | + | Perforated duodenal ulcer. | Meckel's diverticulum. | Gastric mucosa. | Recovered | |
| 67. Klingenhaden[38] | 1936 | 3½ | M | + | + | − | Ulcer of Meckel's diverticulum. | Ulcer at base of a Meckel's diverticulum. | Gastric mucosa. | Recovered | |
| 68. Bunne[9] | 1936 | 7 mo | M | − | + | − | Rectal bleeding of undetermined etiology. | Ulcer of a Meckel's diverticulum. | Gastric mucosa. | Recovered | |
| 69. Bunne[9] | 1936 | 3½ | F | + | + | − | Ulcer of Meckel's diverticulum. | Ulcer of a Meckel's diverticulum. | Gastric mucosa. | Recovered | |
| 70. Thompson[59] | 1937 | 14 | M | + | + | + | Acute appendicitis. | Intramesenteric diverticulum. | Ulcer of gastric mucosa. | Recovered | Diagnosis of Henoch purpura two years previously. |
| 71. Thompson[59] | 1937 | 8 | F | − | + | − | Ulcer of Meckel's diverticulum. | Ulcer of Meckel's diverticulum. | Gastric mucosa. | Recovered | |
| 72. Mallory[44] | 1937 | 7 | F | + | + | + | Meckel's diverticulum with ulcer. | Ulcer of Meckel's diverticulum. | Gastric mucosa. | Recovered | |
| 73. Higgins and Gundy[29] | 1937 | 6 | M | − | + | + | Bleeding Meckel's diverticulum. | Peptic ulcer of Meckel's diverticulum. | Peptic glands. | Recovered | |
| 74. Collins[14] | 1937 | 22 | M | − | + | + | ? | Peptic ulcer in tip of Meckel's diverticulum. | Gastric mucosa. | Died | Hemophilia; poor operative risk |
| 75. Mecray et al.[46] | 1937 | 4 | M | + | − | + | Acute appendicitis. | Perforated ulcer of Meckel's diverticulum. | Gastric mucosa. | Recovered | |
| 76. Gabriel[21] | 1937 | 22 | M | − | + | − | Meckel's diverticulum. | Peptic ulcer of Meckel's diverticulum. | Gastric and pancreatic cells. | Recovered | |
| 77. Matt and Timpone | 1937 | 16 | F | + | + | − | Bleeding peptic ulcer of a Meckel's diverticulum. | Peptic ulcer of Meckel's diverticulum. | Eroded small vessel; gastric mucosa. | Recovered | |

N. B.: only histologically proven cases have been included in this summary.

it must only be considered as a minor component of the symptom-complex.

The treatment of peptic ulcer of a Meckel's diverticulum is surgical. The mortality rate following operation is approximately 4 per cent while the mortality following operation after the ulcer has ruptured is considerably over 50 per cent. Only the autopsy room sees those cases of peptic ulcer of Meckel's diverticulum which have been undiagnosed.

If the patient is markedly anemic, laparotomy should be deferred until one or more blood transfusions have decreased the risk of operative shock. Spectacular changes in the hemoglobin concentration can follow the administration of even a single transfusion. In our case the hemoglobin percentage rose from 46 per cent to 83 per cent nine days later.

If a reasonable doubt exists whether the condition is ulcer of the upper or lower intestinal tract, a gastrointestinal series can be done; however, only the first series of plates delineating the shape of the stomach and duodenum are necessary for the purpose of differential diagnosis.

The patient should receive an extremely restricted diet if he is to be observed or prepared for operation over a period of several days or more. The delay should be as short as possible because of the possibility of rupture of the ulcer during this period.

For reasons which we have stated previously, ileac resection is preferable to simple diverticulectomy followed by some type of purse-string suture. The type of anastomosis may depend only upon the individual preference of the surgeon. We prefer lateral anastomosis. Likewise, whether or not temporary proximal ileostomy is done remains at the discretion of the operator.

The postoperative care is obviously that of any intestinal anastomosis with the possible exception that more blood may be given by transfusion.

## CONCLUSIONS

Lesions of a Meckel's diverticulum are present sufficiently often that the possibility of their presence should be considered in every case presenting unusual gastrointestinal symptoms. Happily an examination of the number of reported cases appearing from year to year shows that this dangerous anomaly is becoming more frequently diagnosed and treated before perforation has forced the puzzled surgeon to perform an "exploratory laparotomy."

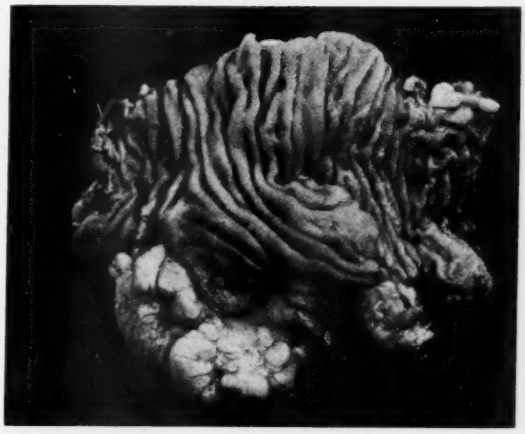

Fig. 1. Segment of ileum with diverticulum everted to show the contrasting mucosa.

Although a routine examination for a possible diverticulum can be quickly and easily made during any laparotomy we do not agree with Greenblatt et al. in that it should be removed, if present, unless the patient has exhibited signs or symptoms referable to the anomaly or if the diverticulum shows upon inspection evidence of being the site of one of its forms of pathology.

The presence of the hemorrhage-age-pain trilogy should point to the diagnosis of diverticular peptic ulcer as certainly as pain-vomiting-fever-leucocytosis indicates an appendicular lesion. The history of hemorrhage alone, if of the sudden, massive, recurrent type, and even if absolutely painless, should suffice to require the positive exclusion of a lesion of Meckel's diverticulum.

## SUMMARY

1. Uncomplicated Meckel's diverticulum occurs in from 1.5 to 3 per cent of all persons. Approximately 25 per cent of

these contain heterotopic tissue which possesses the potentiality of becoming ulcerated.

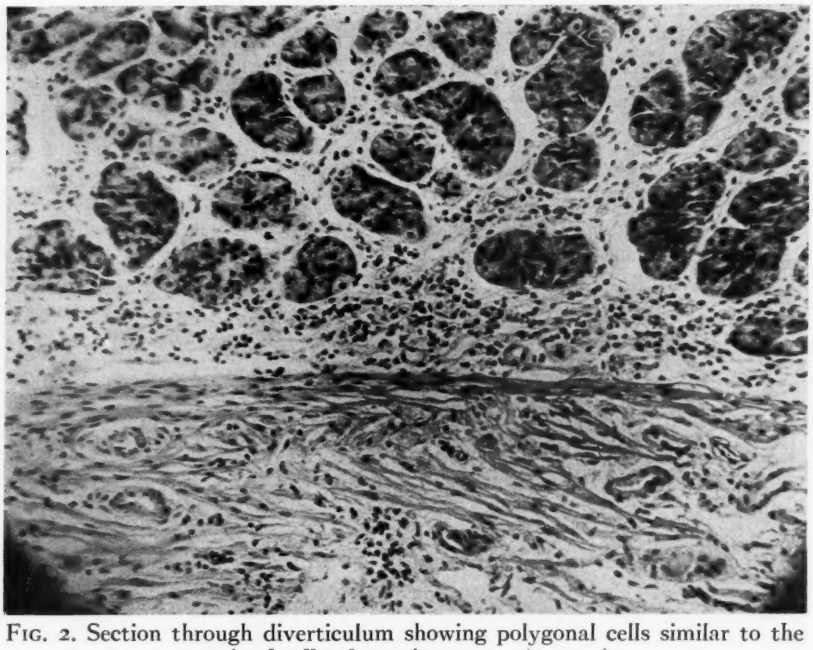

Fig. 2. Section through diverticulum showing polygonal cells similar to the parietal cells of gastric stroma. (× 200.)

2. Theories explaining the presence of heterotopic tissue are stated. The dysembryoma theory apparently satisfactorily explains the conditions found in lesions of Meckel's diverticulum.

3. The pathologic classification of lesions of Meckel's diverticulum as proposed by Greenblatt and his associates is charted. The possibility of clinical diagnosis of lesions of the umbilical and peptic groups is illustrated.

4. The diverticular ulcer trilogy of hemorrhage-age-pain is stressed and evidence supporting their reliability is presented.

5. A review of the literature to 1938, to which the authors add a case diagnosed correctly preoperatively, is presented.

6. Diverticulo-ileac resection following adequate preoperative preparation of the patient, offers an almost perfect surgical record.

## CASE REPORT

I. D., a 16 year old Italian girl, was admitted to the medical service of Dr. Joseph W. Diamond March 25, 1937, complaining of sudden massive bleeding from the rectum. Two days previously she had developed a dull cramp-like pain in the left lower quadrant and on the same evening she passed a large quantity of dark red blood and clots which contained no mucus. On the day before admission to the hospital she had seven bowel movements all of which were composed of pure blood. She had no tenesmus or griping and the abdominal pain had diminished in severity since the time of onset. She had no hemorrhage elsewhere and no petechiae had been noticed. She stated that she had had similar attacks four times during the past three years, the last having occurred eleven months before; since then she had been in good health and her stools had been normal. She had a good appetite, could eat any type of food and never had any symptoms referable to the gastrointestinal tract. She had had the usual diseases of childhood, but otherwise her past history was negative. She began to menstruate at the age of 12, her menses appearing regularly every thirty days and lasting five days. Her last menstrual period began March 5, 1937 and was not unusual.

The patient was well developed, well nourished, but markedly anemic. She had no elevation of temperature, and her pulse and respirations were normal.

The sclerae were extremely pale, as were the lips and buccal mucosa; the nasal mucosa was

slightly inflamed and covered with a scanty mucopurulent exudate. No hemorrhagic manifestations were found in the nasopharyngeal or buccal mucosa.

The chest and heart were normal. The blood pressure was 72/42, the pulse 78 and respirations were 18 per minute.

The abdomen was flat and unscarred. The liver was not palpable, but the spleen descended two fingers below the costal margin during deep inspiration (?) and was slightly tender. No other masses were palpable, but deep pressure over the left lower quadrant caused slight tenderness. Rectal examinations, digital and visual, were negative save for the presence of blood on the mucosa of the rectum and sigmoid colon. The extremities were normal. Neurologic examination yielded normal responses.

Radiographic examination of the gastrointestinal tract following a barium meal showed no evidence of any organic pathology; examination following a barium enema was also negative.

The red blood cell count was 2,740,000 per cu. mm. with a hemoglobin concentration of 46 per cent and a color index of 0.85 per cent. There was marked anisocytosis, moderate piokilocytosis, and slight polychromatophilia. There were 210,000 platelets per cu. mm. of blood. The leucocyte count was 11,700, of which 80 per cent were polymorphonuclear neutrophiles, 1 per cent eosinophiles and 19 per cent small lymphocytes. Bleeding time and clotting time were normal and clot retraction was normal, yielding a firm clot. The capillary resistance test was negative. Blood Wassermann was negative. There was 10.3 mg. of calcium and 3.8 mg. of phosphorus per 100 c.c. of blood. Urinalysis was essentially negative and examination of a stool specimen composed almost entirely of blood was otherwise negative.

The possibility of ulcer of a Meckel's diverticulum was suggested by Dr. M. B. Genaur and diverticulectomy was decided upon. A transfusion of 500 c.c. of whole blood was given by the Scannell method and the patient was placed on massive doses of iron and liver and given a light low residue diet.

On April 10 the erythrocyte count was 4,570,000 with a hemoglobin concentration of 83 per cent. The patient was transferred to the surgical service and three days later laparotomy was performed. The abdomen was opened through a right paramedian incision and a diverticulum was encountered on the anti-mesenteric surface of the ileum approximately 14 inches proximal to the ileocecal junction. A segment of ileum 4 cm. long and containing the diverticulum was removed and lateral anastomosis was done. A temporary ileostomy was made proximal to the line of anastomosis and the abdomen closed following removal of an apparently chronically inflamed appendix.

The postoperative course of the patient was uneventful. She expelled the intraluminal portion of the ileostomy tube on the eleventh postoperative day and was discharged cured fifteen days following operation. At the present time she is in excellent health and is enjoying a normal life in every respect.

The pathologic specimens represented part of the ileum, measuring 9.5 cm. in length. At the mid-portion, a large cherry-sized bulging diverticulum was present. The serosa was shiny throughout. The mesenteric fat extended to the diverticulum and showed a few petechial hemorrhages, while the mucosa of the ileum was covered with bloody mucus. A probe passed easily into the diverticulum whose opening was 20 mm. in diameter. At the base a punched out defect 4 mm. wide was covered by blood clot. The diverticulum itself was filled with mucus and its mucosa was markedly polypous.

Section through the diverticulum showed the mucosa lined by mucus cells with abundant mucus formation. These cells formed ramifying gland-like structures dipping deeply into the stroma. In the deeper layer of the stroma the glands were lined by large polygonal cells with abundant eosinophilic protoplasm with the typical appearance of parietal cells of the stomach. In the basal portions of the glands there were also cells of granular protoplasm similar to septic cells of the stomach. Section through the small intestine showed the typical villi and mucosa of the ileum.

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Wounds of the abdomen which are inflicted by the modern missiles of warfare exert their lethal effect by producing hemorrhage or by engendering sepsis, either through infection carried in with the fragment of high explosive or through damage to the hollow viscera of the belly, especially of the gastro-intestinal tract. The bowel is that portion of the alimentary tube most liable to be wounded, and is damaged nine or ten times as often as is the stomach.

From—"War Wounds and Air Raid Casualties" (Lewis).

# PTOSIS OF CECUM AND ASCENDING COLON

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SEATTLE

## DEFINITION

PTOSIS may be defined as a prolapse downward of one or more of the abdominal viscera to such an extent as to change materially the normal relationship to one another and to the adjacent structures, especially when the subject is in the erect posture.

## ETIOLOGY

1. Predisposing causes:
   (*a*) Arrested development
   (*b*) Maldevelopment
   (*c*) Skeletal posture, shape of abdominal cavity, strength of abdominal wall
2. Exciting causes:
   (*a*) Intra-abdominal pressure
   (*b*) Occupation

To understand maldevelopments it is essential to have at least a rudimentary knowledge of normal development. The alimentary tract is developed from the entodermic vesicle of the zygote.[2] This vesicle becomes divided into an intra-embryonic and an extra-embryonic portion. The intra-embryonic portion becomes the alimentary tract and appendages. About the fifth week the alimentary tract can be differentiated into three main subdivisions, the foregut, the midgut and the hindgut. The divisions are made on form, blood supply, and function. Each part has a separate blood supply. The foregut forms the gastrointestinal tract below the ampulla of Vater to the distal portion of the transverse colon with its circulation supplied by the superior mesenteric artery. The hindgut is caudal to the transverse colon and is supplied by the inferior mesenteric artery. The function of the foregut is digestive; of the midgut absorptive; and of the hindgut excretory. Developmental error is common in the midgut while in the foregut and hindgut malformation is of rare occurrence.

The vitelline duct ordinarily becomes detached about the fourth week of life. The rapid increase of the size of the liver of the midgut is out of proportion with that of the rest of the abdomen. The peritoneal cavity cannot hold all the structures, and the midgut is therefore pushed out through the umbilicus, producing a physiologic umbilical hernia. The apex of the herniated loop is at the former attachment of the vitello-intestinal duct and the termination of the superior mesenteric artery, which originally was the right omphalomesenteric artery. The artery lies in the mesentery and sends branches to the anterior and posterior segments. The mesentery of the herniated loop Dott divides into a pre-arterial and a post-arterial portion. The gut in this stage occupies a sagittal plane. The rotation of the midgut occurs between the fifth and tenth week and may be divided into three stages. In the first stage the pre-arterial segment is on the right side and the post-arterial on the left. The rotation is necessitated by the enlargement and the descent of the liver and pressure of the umbilical artery. The pre-arterial portion and the mesentery become disproportionately long. An enlargement in the post-arterial segment can be seen in an embryo from twenty-seven to thirty days old. This is the beginning of the development of the cecum and appendix.

The second stage of rotation begins about the beginning of the tenth week when the gut returns to the abdomen. Mall suggests that the increase in the length of the loops and their rotation in the abdominal cavity produce enough traction to replace the extra-abdominal intestinal loops within the abdomen. The

internal orifice of the umbilical canal is comparatively small, making it impossible for the entire contents of the hernia to return to the abdominal cavity en masse. The greater size of the cecum may offer sufficient resistance to retain it in the hernial sac, thus permitting the pre-arterial segment to return first. In doing so, it passes behind the superior mesenteric artery which extends from the aorta to the umbilicus. The small intestine as it enters the abdomen pushes the intra-abdominal small gut and mesentery, which has been occupying the midline, to the left and posterior portion of the abdominal cavity. The cecum is the last to be reduced into the abdominal cavity. It occupies a position in the region of the umbilicus, anterior to the small intestines and superior mesenteric artery and beneath the liver. The cecum continues to descend to lie in the normal adult position in the ileocecal fossa. In the twelfth week the superior mesenteric artery, which is brought to the iliac region by the migration of the cecum, lies in intimate contact with the posterior parietal peritoneum and becomes adherent from above downwards to the posterior abdominal wall. Along the right side of the artery the ascending colon and cecum become fixed. The post-arterial mesenteric segment persists as the mesocolon. The pre-arterial segment remains free as the mesentery of the small intestine. The mesentery of the hindgut becomes obliterated by fusion along the midline with the posterior parietal peritoneum.

### DEVELOPMENTAL ANOMALIES

Malrotation and non-rotation depend upon the sequence with which the intestinal loops return to the abdomen. As a result of non-rotation the small intestine lies chiefly to the right. The duodenum passes to the right of the superior mesentery artery. The colon is located entirely on the left side and does not cross the superior mesenteric artery. Normal fixation or absence of fixation, is frequently encountered, producing an abnormally mobile sigmoid or cecum. Non-rotation is the result of a return of the cecum and the post-arterial segment to the abdominal cavity before return of the pre-arterial segment.

Reverse rotation may take place but it is of little significance clinically, because the various parts of the intestinal tract have normal relationships, except that they are in reverse. In malrotation of the midgut, the pre-arterial segment of the midgut passes in front of the superior mesenteric artery and the posterior segment including the cecum also passes in front of the origin of the artery. The mesentery does not become adherent to the posterior abdominal wall. The duodenum and ileum lie side by side in front of the superior mesenteric vessels.

The various congenital anomalies are due to the variation in the size of the embryonic umbilical orifice. Most clinical anomalies, however, arise from faulty third stage rotation. The third stage rotation consists largely of fusion of the various mesenteries with the posterior parietal peritoneum, making the various parts of the gastrointestinal tract less mobile. Early fixation or deficient fixation with the exception of the elongation of the colon produce definite anomalous conditions. The most important of these, pathologically, are those resulting from deficient fixation of the colon.

The position of the retrocecal appendix can easily be explained if one remembers the descent and fusion of the cecum and ascending colon to the parietal peritoneum. The appendix can be turned back behind the descending cecum, becoming enclosed in the mesocolon with the parietal peritoneum. On the other hand, if the appendix takes a position anterior to the cecum in the descent of the colon it will have a free position in the abdominal cavity.

### EXCITING CAUSES

If the occupations of ptotic people require that they be on their feet and

especially if they have to do lifting, the condition progresses from bad to worse. Through the loss of weight and of the fat supporting the abdominal viscera is almost a negligible one.

Long continued intrathoracic conditions,

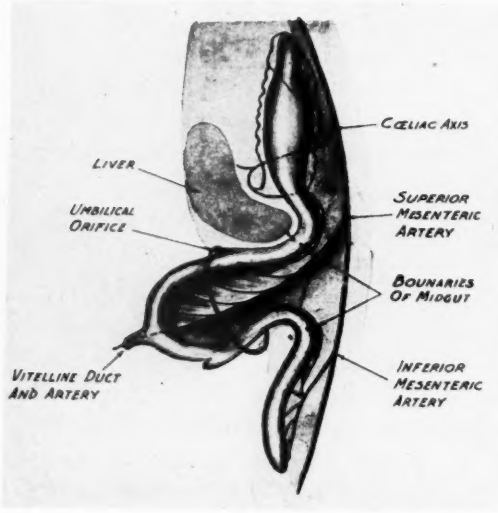


Fig. 1.

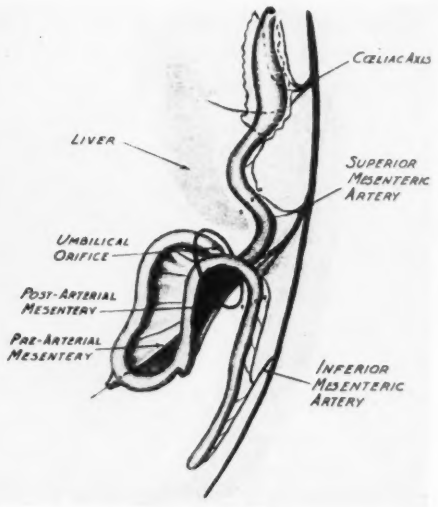


Fig. 2.

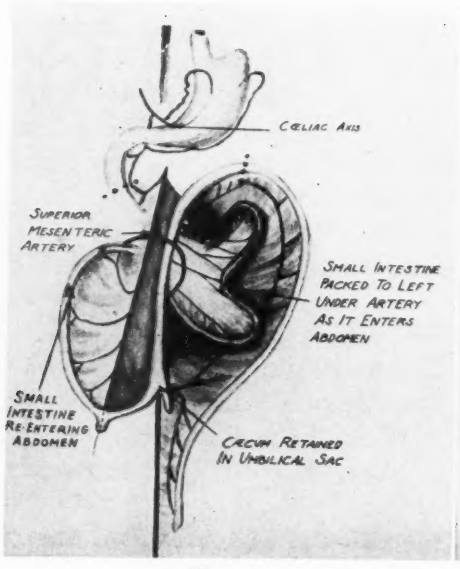
Cœliac Axis
Superior Mesenteric Artery
Small Intestine Packed To Left Under Artery As It Enters Abdomen
Small Intestine Re-Entering Abdomen
Cæcum Retained In Umbilical Sac

Fig. 3.

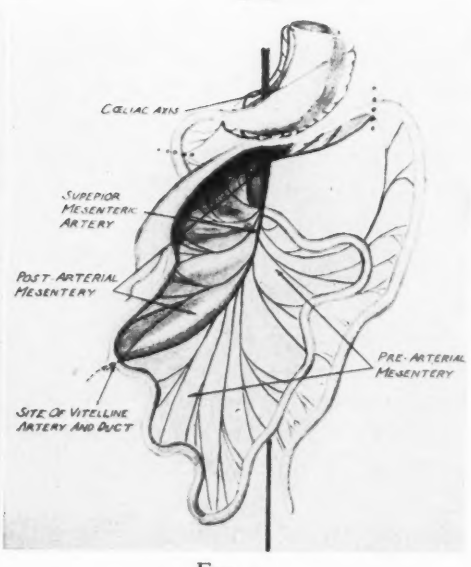


Fig. 4.

Figs. 1 to 4. Stages of development and rotation of the midgut.

in the meshes of the mesentery greater elongation occurs, causing a sagging of the abdominal viscera.

A relaxed abdominal wall after long wasting disease may be followed by a visceroptosis. The relaxation of the lateral muscle groups, that is, the transversalis and the oblique, than that of the recti abdominis, which determines the condition. The part played by the pelvic floor in as large exudates, new growths and, at times, pneumothorax may cause ptosis of the abdominal viscera. In this condition the diaphragm is fixed in the inspiratory position and thus renders its muscular opponents, the muscles of the abdominal wall, useless. Dr. Arthur Keith has made an embryologic and anatomic study of this condition and states that the abdominal muscles in thoracic respiration act as

opponents to the diaphragm. In a shelf-like manner they support the abdominal viscera during inspiration and aid the diaphragm in the elevation of the ribs and thorax. In relaxation of the lateral muscles of the abdominal wall this normal relationship is lost.

The tendency to ptosis often runs in families, various members showing a phthisical chest and giving a history of ancestral tuberculosis.

### PATHOLOGIC ANATOMY AND PHYSIOLOGY

In the normal position of the cecum and ascending colon in the ileocecal fossa, where proper fusion of the post-arterial segment has taken place, they get support from what Coffey calls the iliopsoas shelf. The center of gravity passes through the ascending colon and cecum, well posterior to the anterior border of the psoas muscle. In deficient fusion with a long mesocolon the weight of the intestine forces it into the pelvis, causing a gradual elongation of the mesentery. Nature attempts to correct the defect by laying down a membrane which gradually becomes more dense, and often forming bands which become painful when pulled upon by the distended cecum. These bands produce stagnation by kinking the ascending colon in the region of the hepatic flexure.

A child born with a defective prenatal fixation of the abdominal viscera will have gastrointestinal upsets from birth on. After adolescence, with the greater activity of the child, a gradual development of the typical ptotic figure is the rule. The colon, dropping from its normal location, slides off the psoas shelf into the pelvis. The kidney fossa becomes shallower. The kidney often slides down. The drag on the bottom of the stomach causes displacement of the pyloric end of the stomach, retarding the gastric contents. This causes indigestion with subsequent loss of flesh and further reduces the visceral support by decreasing the intra-abdominal pressure. Gas and fluid in the gastrointestinal tract are increased. The stretching of the gastro-intestinal muscles causes a gradual reduction of peristalsis. The efficiency of digestion is generally lowered. The abdominal organs tend to gravitate more and more to the lower portion of the abdomen and pelvis.

The drag on the diaphragm and the fascia attached to the ribs causes the lower portion of the chest to become narrow and the angle of the ribs with the vertebrae to become sharper. The lower end of the spine and the pelvis are tilted backwards in an effort to hold the load by means of the thoracic fascia.

The normal curvature of the spine disappears, and the spine becomes relatively straight with the lower end tilted relatively forward and upward and the upper part backward.

The drag on the visceral nerves and sympathetic plexus materially affects the nervous stability of the patient, and these individuals are often classified as neurasthenics. The intestinal stasis and resulting autointoxication extending over years causes a depletion of the adrenals with a gradual lowering of the blood pressure, increasing weakness and making for easy exhaustion on physical and mental exertion.

### SYMPTOMATOLOGY

The discussion of symptoms is here limited to those of right sided abdominal ptosis only, the ptosis of the cecum and ascending colon. This condition is, practically without exception, due to a congenital defect, the cecum and ascending colong having failed to fuse properly with the posterior parietal peritoneum. The symptoms generally appear at the time of adolescence and are often diagnosed as intestinal colic. There are attacks of right sided abdominal pain which may last only a short time or may last for days, with rest in bed necessary before they subside. As time goes on the cecum and ascending colon become larger and their contents are less easily expelled.

Nature meanwhile tries to come to the rescue by laying down supporting mem-

branes along the line of stress from the right lateral wall of the abdominal cavity to the cecum and ascending colon. These

| CASE | PAIN | VOM. | CONST. | DIAR. | TOX. | DEF. ABD. | AB. MASS | TREAT. | RESULT | MORT. |
|---|---|---|---|---|---|---|---|---|---|---|
| G.S. 20 Male | 6 Yrs. Periodical | + | + | | + | | | App. Colop. | Cured | 0 |
| A.N. 28 Male | 7 Yrs. | + | +++ | | ++++ | | | App. Colop. | Cured | 0 |
| M.A. 22 F. | 10 Yrs. | + | +++ | + | ++ | ++ | | App. Colop. | Cured | 0 |
| M.R. 26 F. | 18 Yrs. | ++ | ++++ | | +++ | +++ | ADH. Colitis | Free Adh. Colop. | Satisf. Imp. | 0 |
| W.G. 34 Male | For Many Years | + | ++ | | + | + - | Acute Append. | App. Colop. | Cured | 0 |
| M.C. 20 F. | 7 Yrs. | +++ | ++ | + | ++ | + | | Colop. Free Adh. | Cured | 0 |
| A.S. 20 F. | Since Colic Appd. | +++ | + | | ++ | ++ | Gen. Pelvic Colitis | App. Adhesive Colop. | Clin. Cured | 0 |
| W.S. 28 F. | 10 Yrs. | | +++ | | +++ | ++ | Colitis ++ | App. Colop | Cured | 0 |
| C.F. 42 F. | 25 Yrs. Periodical | + | ++ | + | ++ | ++ | Colitis ++ | Colop. | Imp. | 0 |
| C.T. 7 F. | 4 Yrs. | + | ++ | | +++ | | | App. Colop. | Cured | 0 |
| S. 33 F. | 6 Yrs. | + | +++ | + | ++ | + | | App. Colop. | Cured | 0 |
| E.P. 22 F. | 6 To 9 Years | + | +++ | | ++ | + | Acute Append. | App. Colop. | Cured | 0 |

CURED.....75% IMPROVED.....25% MORTALITY.....0%

FIG. 5. Tabulation of cases with prominent symptoms, treatment and result.

membranes stand out as dense white bands, often kinking the bowel in the region of the hepatic flexure, still further impeding the movements of the intestinal contents. The appendix in these cases is apt to become infected because of the stagnation in the colon. Removal of the appendix gives little or no relief.

There is vomiting after the pain has started, not at the beginning as in an attack of acute appendicitis. There may be fever of a degree or so if the stasis is considerable.

On physical examination we often find a palpable cecum and an ascending colon which contains gas and fluid. There is tenderness along the attachment of the membranous bands. In severe cases we find a ptotic figure and signs of neurasthenia.

A barium meal shows a low lying cecum and often a retention of barium for days. A barium enema will show the same and, if there are tight bands, delay of the barium enema at the region of the hepatic flexure can be seen.

## DIAGNOSIS

In the differential diagnosis we may consider all right sided abdominal conditions which give rise to pain, vomiting and toxemia, as well as right sided kidney lesions and, in women, right pelvic lesions. The succussion splash in the cecum is said to be pathognomonic of a redundant cecum with stasis. Gastrointestinal *x*-ray studies are conclusive, but a diagnosis can generally be made without the aid of the *x*-ray.

The history of right sided abdominal pain from the time of adolescence which is apt to get worse on physical exertion especially when the patient has to be on his feet for any length of time is characteristic. This pain may become worse with each attack. Pain occurring during or after an evening of dancing is very suggestive. The location of the pain is higher than the average appendix. It is described as a dragging or a heavy pain in the right side or, if partial obstruction has occurred, as colicky.

## PROGNOSIS

Due to the stasis and irritation produced by decomposition a colitis is set up which in advanced cases spreads from the ptotic portion of the colon. The symptoms are thus far in excess of what one would expect from the original pathology. After the correction of the pathologic anatomy the colitis must be treated. Otherwise, the prognosis is good.

## TREATMENT

Medical management consists in relieving the intestinal stasis and attempting to shorten the mesentery by deposition of fat in the mesenteric meshes. The patient should be kept in bed with the foot of the bed raised and put on forced feeding, sometimes for months. A gain in weight may relieve all symptoms. The shortened mesentery supports the intestines well enough to prevent kinking. If the weight can be maintained after a return to usual activities, good health may continue, but generally a gradual loss of weight causes return of the symptom complex.

Belts and abdominal supports may be valuable in gastroptosis and general ptosis

but I believe they are worse than useless in right sided ptosis, except for their psychic effect. Surgical correction should be carried out before a marked colitis has set in.

Surgical treatment is in most cases successful. Preoperative management requires emptying of the colon with a mild laxative and enemata. Hospitalization for some days prior to the operation is advisable. Intraperitoneal vaccination is also valuable, especially if there is concomitant colitis. In debilitated patients, a build up program before operation should be carried out. Transfusion is helpful.

Spinal anesthesia gives excellent relaxation and should allow sufficient time. The operation can be performed in less than an hour. Ether anesthesia, where there is no contraindication, is satisfactory.

A right pararectus incision is made, of sufficient length to enable one to expose the hepatic flexure and the cecum. The bands and adhesions are released and the appendix is removed. An incision is made lateral to the cecum and ascending colon as high as the hepatic flexure, this being the lateral leaf of the ascending mesocolon. The peritoneum is reflected laterally, exposing the perirenal fat, practically a bloodless dissection. Care is taken not to injure the ureter.

Sutures of chromic catgut or dulax are passed through the edge of the reflected peritoneal fold, the first through the lower end of the reflected fold and the region of the appendiceal stump. The following sutures are inserted ½ inch to 1 inch apart through the anterior longitudinal band of the cecum and ascending colon. I have found it advantageous to leave the sutures untied until they are all inserted. When the sutures are tied the pendulous cecum and ascending colon roll into the denuded space where they soon will become firmly attached. As there generally is a coexisting ptosis of the right kidney, the kidney will become fixed in its proper position. At times the bands and membranes which have been formed in nature's attempt to relieve the pathologic condition can be used to strengthen the anchoring of the ascending colon. If there still remains a sharp kinking at the hepatic flexure the incision can be curved around and the condition corrected by suitably placed sutures.

In cases where there is a long transverse colon which falls down into the pelvis, dragging on the hepatic flexure, I anchor it to the anterior abdominal wall medial to the hepatic flexure. Sutures are inserted through the root of the omentum and parietal peritoneum of the anterior abdominal wall. This is what Coffey describes as his hammock operation. It can be extended right across the abdominal cavity if necessary and will give support to a ptotic stomach.

These patients have remarkable little postoperative disturbance. The relief of the partial obstruction and the absence of intestinal toxemia give them a feeling of well being. They can leave the hospital when the wounds are healed and are permitted to resume active life as after any other similar abdominal operation. Colitis, if present, is treated by diet and other measures. The patient often expects to be completely cured and does not coöperate for a long enough time to have the colon return to normal. In cases where a portion of the colon is much involved it might be wise to do a partial colectomy.

## CASE REPORTS

I have operated on twelve patients and have seen a number of other cases in which no operation has been done, but in which the diagnosis has been established by radiographic study. The twelve operations have produced highly satisfactory results, with ten complete clinical cures and two much improved. The colitis which existed in the latter two cases necessitates a more or less strict dietary regime. They are, however, relieved of the toxemia and the symptoms associated with the partial obstruction. Typical case histories are appended.

Case I. A. S., male, age 20, complained of right sided abdominal pain occurring at intervals of a few weeks, generally following dietary indiscretion or excessive physical exertion. The symptoms first appeared at the age of 14 and had since gradually increased in severity. Vomiting occurred with the attacks, but rarely fever. The pain lasted for two or three days. A diagnosis of appendicitis had been made.

There was a palpable cecum containing gas and fluid. The leucocyte count was 10,000, the red count 4,400,000. A barium meal revealed a low lying cecum with stasis and probable kinking at the hepatic flexure.

At operation the appendix was found to be normal, but there was a well developed Jackson's membrane with tight bands at the hepatic flexure. The cecum and ascending colon were distended and pendulous. The appendix was removed and a Waugh operation was performed. The postoperative course was uneventful and complete recovery followed.

Case II. A. N., male, age 28, had pain in the abdomen especially in the right side, fever, vomiting, constipation, attacks of diarrhea, and a rash which he had been told might be due to syphilis.

There was a palpable mass in the right side of the abdomen, movable from side to side, and containing fluid and gas. The temperature was 103°, the Wassermann negative, leucocytes 12,000. The urine was normal. A diagnosis of intestinal stasis with toxemia was made. The intestinal stasis was relieved, the symptoms disappeared, and the patient returned to work.

One month later the condition recurred, and the patient was operated on. A large and pendulous cecum and ascending colon were found with bands between the ascending colon and the lateral wall of the abdomen. The appendix showed no gross changes. The appendix was removed and a Waugh operation performed. Smooth recovery ensued, with no return of symptoms.

Case III. N. A., female, age 25, had had periodic right sided abdominal pain for the previous ten years. It was apt to come on after exertion, such as dancing, but disappeared overnight or in a day or two. She was easily fatigued, had frequent headaches and was physically below par.

Gas and fluid could be made out in the cecum and ascending colon. There was tenderness along the ascending colon. A barium meal revealed a pendulous cecum with delayed emptying of the cecum and ascending colon. Tight bands at the hepatic flexure were found at operation. The appendix was apparently normal. It was removed, the bands cut, and a Waugh operation performed. Convalescence was uneventful and recovery complete.

Case IV. H. P., female, age 25, had abdominal pains from 12 years of age. An appendectomy was done at the age of 20, when she was told that the appendix was normal but there was some pelvic condition causing her trouble ("fibroid uterus"). Two years later she was operated on for an ovarian cyst. The right sided abdominal pain with constipation and toxemia persisted, however, and sometimes confined her to bed for days at a time. General debility and neurasthenia were noted. Gastrointestinal radiographic study revealed stasis in the cecum and ascending colon. A long pendulous cecum rested on the pelvic floor. There were probably adhesions from the previous operation. The transverse colon was long and occupied the pelvis. The colon was kinked at the hepatic flexure.

A long pararectus incision was made on the right side. There were adhesions between the cecum and the old appendectomy scar, as well as bands and membranes between the cecum and ascending colon and the right side of the abdominal wall. The colon was inflamed and edematous. The adhesions were freed and colopexy performed with the incision curving around the hepatic flexure. Sutures were placed in the root of the large omentum and to the anterior abdominal wall medial to the hepatic flexure halfway across the abdomen.

In this case there was marked colitis extending practically half way across the transverse colon and very marked at the hepatic flexure. The patient's postoperative course was smooth, a diet had to be followed. However, the stasis was relieved and if there is no dietary indiscretion the patient's health is good.

Case V. W. G., male, age 34, had periodic abdominal pain for several years which at times forced him to bed. He felt better when he kept his bowels moving with laxatives and enemata. He was brought to the hospital as an emergency in the middle of the night with a fever of 103, marked tenderness and rigidity over the right side of the abdomen. Tenderness was equally marked under the right costal

margins and over the right lower quadrant. Leucocytes numbered 18,000, with 90 per cent polymorphonuclears.

A long right pararectus incision made. An acutely inflamed appendix with a distended cecum and ascending colon and tight bands at the hepatic flexure causing almost complete obstruction were observed. The ascending colon was inflamed. The appendix was removed, the bands cut and a modified right sided colopexy done. A stormy convalescence for the first few days was followed by complete recovery.

Case vi. M. C., female, age 20, had had pain since the age of 13 or 14 which had gradually grown worse. She was afraid to go to dances because of the pain which followed. The attacks were always associated with vomiting, rarely by fever. There had been considerable loss of weight. One year before an appendectomy had been performed, but a long convalescence had been followed by no improvement.

There was a palpable cecum and ascending colon, tenderness over the right side of the ascending colon. Blood and urinary findings were normal. Radiographic study showed six hour retention in the stomach, a large pendulous cecum and colon, and apparently bands at the hepatic flexure.

Operation confirmed the findings of the radiographic study. Adhesions between the pylorus, duodenum and hepatic flexure and Jackson's membrane were noted. The adhesions were released and a colopexy performed. Smooth convalescence followed.

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# MESENTERIC VASCULAR DISEASE*

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THE increasing incidence of vascular disease[1] makes a better knowledge of the abdominal manifestations desirable. Recently a number of significant contributions to this subject have been made.[2,7]

In the present paper the etiology and pathology of mesenteric vascular disease are reviewed briefly, the clinical manifestations are discussed in detail and certain points in the surgical management of these cases are outlined.

The etiologic factors involved in vascular disease of the mesentery and the relative frequency of these factors as observed in the Peter Bent Brigham Hospital are shown in Tables I and II. The important fact to be noted is that arterial thrombosis accounted for over 50 per cent of the cases. This means that, except for arteriosclerosis, an obvious clinical cause for the occlusion such as a source for an embolus, cirrhosis of the liver or intra-abdominal sepsis was lacking. Moreover, in those cases in which the patient's condition was most favorable for surgical therapy, generalized arteriosclerosis was not a prominent finding. Consequently, an obvious etiologic factor should not be considered essential in the clinical diagnosis of mesenteric vascular disease.

The mechanism and pathologic anatomy of mesenteric vascular occlusion merits brief consideration. Klein's[8] careful study of the available experimental and pathologic material has shown that the closure of a mesenteric artery may be followed by three possible results, depending largely upon the site and the rapidity of onset of the occlusion and on the status of the collateral circulation. These possibilities are: (1) complete establishment of a collateral circulation which may (a) persist effectively throughout the life of the patient, or (b) subsequently break down, usually due to a progression of the occlusive process or cardiac failure; (2) intestinal symptoms, such as pain, vomiting, or obstipation due to a blood supply which is sufficient for life of the parts, but not for function; or (3) intestinal infarction with the injury varying from a moderate degree of necrosis to gangrene of the entire intestinal wall.

If a sudden occlusion occurs at the base of the superior mesenteric artery, an extensive anemic infarction of the bowel results. However, if the occlusion is sufficiently gradual in onset a completely competent collateral circulation may develop. If the occlusion occurs in a branch of the superior mesenteric artery, anywhere above the last arcade, there may be no symptoms if the occlusion is gradual in onset, but if, as usually happens, the occlusion occurs suddenly there is an associated vascular spasm of the collateral circulation and an anemic infarction of a portion of the bowel results. However, in this location although the initial infarction is anemic in character it subsequently becomes blood soaked and hemorrhagic, because as the vascular spasm subsides, there is a back flow of blood from the collateral circulation into the involved area. As the majority of arterial occlusions are in this location, one usually finds at operation or necropsy an edematous, swollen, hemorrhagic bowel. Occlusions below the last arcade in most cases probably cause no symptoms. In certain instances transient symptoms, falling into the group of "so-called abdominal angina" may be observed.[4] In venous occlusion there is a continued flow of blood from the arteries into the infarcted area, so that the

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lesion is always of a blood soaked, hemorrhagic nature. Thus, although the mechanism is different, the final gross pathologic picture in both venous and arterial occlusion is quite similar.

Table I

- Arterial
  - (a) Embolic
    1. Rheumatic heart disease with auricular fibrillation.
    2. Arteriosclerotic heart disease with mural thrombi in the heart or aorta.
    3. Acute endocarditis.
    4. Subacute endocarditis.
  - (b) Thrombotic
    1. Arteriosclerosis.
    2. Thrombo-angiitis obliterans.
    3. Syphilis.
- Venous.
  - (a) Intra-abdominal sepsis.
  - (b) Vascular stasis
    1. Cirrhosis of liver.
    2. Other mechanical causes such as carcinoma, enlarged glands, etc.
  - (c) Blood dyscrasias
  - (d) Idiopathic (probably often a mechanical cause, such as a volvulus or internal hernia which has become reduced before operation or autopsy).

Although an occlusion of the mesenteric veins may be gradual in onset, the clinical picture, as will be discussed later, is sometimes as sudden in onset as in an arterial occlusion. There are two reasons for this. First, when the occlusion is extensive, there is a tremendous loss of blood from the arterial side into the involved segment so that profound shock may develop with even more dramatic suddenness than is observed in arterial occlusion. Secondly, in low grade, slowly progressive thrombosis of the mesenteric veins, the compensatory powers of the collateral circulation may be sufficiently great to prevent symptoms up to a certain point and then collapse suddenly.[5] It can also be seen from the above discussion that arterial occlusion may be so small as to produce either very slight or very slowly progressive signs so that, as will be pointed out no fundamental distinguishing features can be expected in the clinical aspects of arterial or venous occlusion.

Table II

| Cause | Type of Occlusion | No. of Cases |
|---|---|---|
| Arteriosclerosis | Arterial thrombotic | 15 |
| Cirrhosis of liver | Venous thrombotic | 3 |
| Idiopathic | Combined | 3 |
| Endocarditis | Arterial embolic | 3 |
| Intra-abdominal sepsis | Venous thrombotic | 2 |
| Rheumatic heart disease | Arterial embolic | 2 |
| Thrombo-angiitis obliterans | Arterial thrombotic | 1 |
| Syphilis | Arterial thrombotic | 1 |

The anatomic distribution of the lesions in the present series is shown in Table III. The ileum alone was involved eight times; the jejunum alone, three times; and both ileum and jejunum, thirteen times. In four cases the lesion involved the colon in addition to the small bowel, and in only two cases was the large bowel alone involved. This distribution is quite typical and is similar to that of previously reported series. The significance of the anatomic distribution lies in the fact that the ileum is involved in over 80 per cent of the cases. This has an important bearing on the surgical approach to the lesion.

Table III
ANATOMIC DISTRIBUTION

| | Arterial | | Venous | | | Total |
|---|---|---|---|---|---|---|
| | Embolic | Thrombotic | Infectious | Vascular Stasis | Idiopathic | |
| Jejunum only | 0 | 3 | 0 | 0 | 0 | 3 |
| Ileum only | 1 | 4 | 0 | 2 | 1 | 8 |
| Jejunum and ileum | 1 | 7 | 2 | 1 | 2 | 13 |
| Jejunum, ileum and ascending colon | 2 | 2 | 0 | 0 | 0 | 4 |
| Ascending colon only | 0 | 1 | 0 | 0 | 0 | 1 |
| Sigmoid colon only | 1 | 0 | 0 | 0 | 0 | 1 |

## CLINICAL MANIFESTATIONS

A correct diagnosis of mesenteric vascular disease is rarely made, partly because of the comparative rarity of the condition

and partly because of certain inherent difficulties in diagnosis. A source of embolus, copious melena, a rapid fall of temperature and severe abdominal pain were at one time thought essential for a diagnosis, and even today experienced clinicians will dismiss the possibility of a vascular accident in the abdomen because "the patient is not sick enough." It cannot be emphasized too strongly that mesenteric vascular occlusion, particularly when it involves the arteries, is not always fatal and the severity and intensity of the symptoms are as variable as those of peripheral or coronary artery disease.

For convenience of discussion, the clinical manifestations of mesenteric vascular disease may be divided into three groups: Those which result from spasm or small non-fatal occlusions of the mesenteric arteries, so-called "abdominal angina" or "abdominal intermittent claudication"; those which follow an extensive occlusion of either a mesenteric artery or vein, "mesenteric vascular occlusion"; and those which are a consequence of the rupture of a sclerotic artery with intraperitoneal hemorrhage, "intra-abdominal apoplexy."

"Abdominal angina" has been described for years, but until recently, a lack of correlation between clinical inference and pathologic confirmation has prevented its acceptance as a clinical fact. Such distinguished clinicians as Sir William Osler and Sir Clifford Allbutt doubted its occurrence. However, sufficient evidence has now been accumulated to prove that vascular disease of the mesentery can cause abdominal pain in the absence of gangrene of the bowel or peritoneal irritation.[4,7,10] The exact mechanism involved in the production of abdominal angina is not definitely established. That it is due to vascular spasm seems less likely than that it is the result of a relative anoxemia from small, non-fatal occlusions. The partially occluded vessels, although sufficient for viability, are unable to meet the increased demands for blood necessary for function, exactly as in the arteriosclerotic leg which is over-exercised.[4]

The clinical importance of abdominal angina lies in the fact that it may be the precursor of extensive mesenteric vascular occlusion, and a proper appreciation of its characteristics is of importance in the early recognition of this highly fatal surgical emergency. In the cases of this type which we have had an opportunity to observe, the pain has been rather deep seated in the mid-abdomen or epigastrium, has not radiated, and has not been well localized. There has been a definite relation between the ingestion of food and the onset of pain and in three instances the patients have abstained voluntarily from heavy eating, with resultant relief. Although severe, the pain has not been associated with spasm or exquisite tenderness of the abdominal wall. Naturally, in such cases, a thorough examination of the biliary and gastrointestinal tracts by means of one *x*-ray is necessary. Then if the findings are negative a diagnosis of abdominal pain of vascular origin is justified.

In a previous communication[4] we have commented on the characteristics of abdominal vascular pain, pointing out that it appears to be a true visceral pain and consequently is not associated with muscular spasm or exquisite tenderness of the abdominal wall. This is of considerable importance in the early diagnosis of extensive mesenteric vascular occlusion, because the pain which occurs in the early stages of this condition differs from simple vascular pain only in its constancy. It also has the characteristics of true visceral pain as it is not associated with muscular spasm or exquisite tenderness until gangrene and peritonitis have been established. This contrast between the severity of the pain and the paucity of the physical findings is one of the most significant diagnostic features in the early stages of mesenteric vascular occlusion.

The extensive occlusion of either a mesenteric artery or vein presents a fairly definite clinical picture. Loop[9] was the first investigator to emphasize distinguishing features. He pointed out that the paramount symptoms are a disturbed function

of the gastrointestinal tract, manifested by severe abdominal pain, vomiting, difficulty in moving the bowels without complete obstruction, and shock of variable degree. He noted the absence of fever, muscular spasm, and distension. Dunphy and Zollinger[2] arrived at similar conclusions from an analysis of four personally observed cases, and made a correct preoperative diagnosis in a fifth case. This led to a successful resection. Since that time at the Peter Bent Brigham Hospital a correct preoperative diagnosis has been made in two more cases and in a third case a correct diagnosis was proved by post-mortem examination, the patient having been moribund from associated diseases.

The features which we have noted are: (1) Regardless of the duration of the attack, the clinical picture has not been typical of the common surgical emergencies. The localized tenderness of appendicitis, the rigidity of perforated ulcer and the visible peristalsis and early distention of mechanical intestinal obstruction are not present. Usually one is able to makes no definite diagnosis. However, the patients always appear ill from an abdominal lesion which simulates, but is not typical of, obstruction. (2) The character of the pain is quite out of proportion to the clinical findings and persists after ordinary measures have been instituted for its relief. (3) The only constant physical finding is deep abdominal tenderness more or less generalized with rebound tenderness referred to the point of pressure. In the later stages the signs of generalized peritonitis develop, but these should not be considered essential in the early diagnosis. (4) The leucocyte count and pulse rate are almost invariably elevated disproportionately to the temperature and other signs. Finally, it is evident that there is a gastrointestinal disturbance, but the manifestations of this are not constant. Difficulty in moving the bowels without complete obstruction or, less commonly, with bouts of diarrhea, is seen in nearly all cases. Vomiting is variable. Bloody diarrhea should not be expected, but when found, it is a valuable contributory sign. Rapidly progressing shock is uncommon and in our experience is more frequently seen in extensive venous occlusion that in arterial occlusion.

We have been able to establish no criteria by which we can distinguish between venous and arterial occlusion other than a fortuitously obvious etiologic factor. Some observers have noted that arterial thrombosis is more rapid in onset with marked toxicity and is rapidly and progressively fatal. This is not invariably the case. In fact, in our experience the most rapidly fatal case was one of extensive thrombosis of the mesenteric veins. Moreover, the experimental observations of Scott and Wangensteen[11] indicate that a venous occlusion, if extensive, should be rapidly fatal because there is a tremendous loss of blood from the arteries into the involved sement of bowel. Warren and Eberhard[5] also have stated that the differential diagnosis between arterial and venous thrombosis is highly speculative.

Hibbard[12] has called attention to the roentgenographic manifestations of experimental mesenteric vascular occlusion. He found that moderately distended loops of bowel, gas shadows, and fluid levels could be detected by means of a plain *x*-ray film taken in the upright position a few hours after the experimental ligation of the mesenteric artery or vein in dogs. These signs developed before there was clinical evidence of obstruction. Although not distinguishing between vascular occlusion and mechanical intestinal obstruction, he felt that this evidence would be of value in establishing an early diagnosis. However, it should be pointed out that while such evidence may be of assistance, a negative Roentgen examination of the bowel is of no diagnostic value. In the present series seven patients had had *x*-ray studies which varied from a plain film of the abdomen to a complete examination of the gastrointestinal tract by means of a barium enema and barium meal. In none of these cases was there conclusive evidence of intestinal obstruction. Consequently, the *x*-ray should

be considered only as a means of furnishing contributory evidence and is not in any sense conclusive.

It should not be maintained that the features which we have outlined are pathognomonic of mesenteric vascular occlusion nor are these essential to diagnosis, for the white blood count, the temperature, and the physical findings may be as misleading in this condition as in appendicitis. However, these features, when present, render a diagnosis much more tenable than is generally realized. The condition which we have had the most difficulty in distinguishing from mesenteric vascular occlusion is a subacute pancreatitis in an elderly patient. However, the temperature tends to be higher in pancreatitis, vomiting is a more prominent symptom and the pain in subacute cases is more easily relieved by sedatives. In the severe cases of pancreatitis, signs of early peritonitis are more striking than in mesenteric vascular occlusion. The blood diastase is of value in the differential diagnosis of doubtful cases.

It may be argued that all cases do not conform to the syndrome which we have outlined. Every clinician of experience can recall cases in which an entirely unsuspected vascular occlusion of the mesentery was revealed at post-mortem. The reason for this lies not in the site, type or degree of the occlusion, but in the underlying etiologic factors or intercurrent disease. Thus, in 30 per cent of the arterial cases in the Peter Bent Brigham Hospital series the presence of extensive coronary artery disease or cerebral arteriosclerosis had rendered the patients moribund before the abdominal vascular accident occurred. Similarly in 60 per cent of the venous group the terminal stages of cirrhosis of the liver or intra-abdominal sepsis were associated with an unrecognizable thrombosis of the mesenteric veins. These cases, however, in no way detract from the value of the syndrome described above in the recognition of early mesenteric vascular occlusion. Given a patient with (1) severe abdominal pain; (2) a paucity of physical findings so that the picture as a whole is confusing and does not fit any of the usual surgical emergencies; (3) a high leucocytosis and high pulse rate with a relatively low temperature; (4) abdominal tenderness more or less generalized; and (5) evidence of a gastrointestinal disturbance manifested by either vomiting or diarrhea or signs of subacute obstruction—mesenteric vascular occlusion should be considered as the most likely cause.

It is not generally realized that one of the rarer manifestations of abdominal vascular disease is intraperitoneal hemorrhage. This condition is the abdominal counterpart of cerebral hemorrhage. Less than a dozen cases have been reported in the literature. Although very rare, it is of sufficient interest to warrant a brief description. The clinical picture is one of severe abdominal pain accompanied by signs of peritoneal irritation, shock and hemorrhage. In the majority of the reported cases the diagnosis has been acute pancreatitis or perforated ulcer. In the only case which we personally have observed[13] the clinical picture was quite suggestive of mesenteric vascular occlusion which was the preoperative diagnosis.

A word must be said in regard to the indications for operative intervention in abdominal vascular disease because, as has been emphasized before, spontaneous recovery may ensue. Actually in the majority of cases by the time the diagnosis has been established, operation is imperative. However, with an increasing interest in the disease, cases are being recognized earlier and there is such a thing as operating too soon, because (1) the disease may not progress to gangrene and (2) even if gangrene is imminent a very early operation may find the lines of demarcation poorly defined. This situation is particularly likely to arise in the case of a patient with auricular fibrillation already in the hospital for medical care. Following a sudden change from fibrillation to a normal rhythm the patient may show evidence of a small embolus to the brain or extremity and then suddenly present the picture of an abdominal castastrophe. There may be severe abdominal pain, vomiting, and

shock of considerable degree. The diagnosis of a vascular accident in the abdomen is obvious and the tendency is to operate at once. We feel that in such cases one should treat the patient for shock for a period of at least several hours, noting at frequent intervals the blood pressure, pulse rate, leucocyte count and temperature. Careful repeated examinations of the abdomen are essential. A steadily rising pulse rate and leucocyte count and increasing abdominal tenderness, even without evidence of marked rigidity or abdominal distention, are indications for surgery. If, however, the white count rises only slightly, the pulse rate tends to stabilize and no abnormal physical findings are noted, a conservative policy is indicated and recovery may be expected.

### SURGICAL MANAGEMENT

Once operative intervention is elected there is no place for conservative measures in the treatment of abdominal vascular disease. Enterostomy, sometimes a life-saving procedure in intestinal obstruction, is worthless once gangrene has been established. Exteriorization is also fraught with danger because if the thrombosis is venous, there is a tremendous loss of blood into the involved segment of bowel. Moreover, even in arterial occlusion the removal of the gangrenous segment of bowel is accompanied by a striking immediate improvement in the patient's condition, as if there had been absorption of toxic products from the gangrenous loop.

As has been emphasized many times before, the poor general condition of these patients is no argument against resection since the alternative is a fatal issue. There is but little trauma to a well conducted resection and it is amazing how well these patients survive an apparently hopeless situation. Thus, recovery has ensued in a case in which no anesthesia was necessary because of the moribund state of the patient[14] and in nearly every case the patients have seemed to be in better condition immediately after than immediately before the operation. This striking improvement is undoubtedly directly related to the complete removal of the gangrenous bowel.

Although the necessity for resection is well established, the proper treatment of the divided loops of bowel is a debatable point. An immediate anastomosis, as recommended by Elliot,[15] is obviously the ideal procedure. However, in his successful case Elliot performed a secondary closure. Jackson, Porter, and Quinby[16] cautioned against immediate suture because of the difficulty in determining lines of demarcation and the danger of advancing gangrene. More recently a multiple stage method has been advised by Green and Allen.[17] It seems to us, however, that each case must be handled as an individual problem. If the lines of demarcation are well defined and the condition of the patient is satisfactory a primary anastomosis can be done. On the other hand, exteriorization of the divided loops may be the procedure of choice in the face of poorly demarcated necrosis or a badly reacting patient. It is worth noting that in a study of the reported operative cases of the fourteen years between 1920 and 1934 the highest percentage of recoveries was found to occur in the group in which resection with immediate anastomosis was done.[2]

In the operative approach a generous right rectus incision should be used and the cecum and terminal ileum at once identified. The extent of involvement of the colon should be determined and then the small bowel explored proximally, delivering the involved segments at the same time. When the proximal border of the gangrene is found, the entire extent of the lesion is known and the resection can at once be begun. If the terminal ileum is not involved, exploration from this point proximally should be done as the colon and upper jejunum are rarely involved. If the exploration is systematically done in this order very little time is lost in determining the extent of the lesion and a good start towards a well conducted resection is obtained.

There are several points to be noted in the surgical management of intra-abdominal apoplexy. If the diagnosis is suspected a rather high right rectus incision should be used. It is more likely, however, that the surgeon will encounter an abdomen full of blood in an elderly individual in whom the diagnosis has not been suspected. In such cases the gastrohepatic ligament should be investigated first as it is the most frequent source of hemorrhage. If the ligament is swollen and thickened by active hemorrhage, it should be opened and the bleeding vessel identified and ligated.[18] However, if all signs of continued hemorrhage have ceased, it may be permissible to leave the area undisturbed without identifying and ligating the precise bleeding point.[13] If the gastrohepatic ligament is not involved and the gastric vessels appear normal along the greater curvature, the superior mesenteric artery should be investigated. This is the next most common site for intraperitoneal hemorrhage in abdominal vascular disease.

## SUMMARY

1. The etiologic factors involved in mesenteric vascular disease are not sufficiently obvious clinically to be considered essential in the diagnosis of this condition.

2. In the majority of cases the gross pathologic anatomy of arterial and venous mesenteric vascular occlusions is quite similar. The pathogenesis of each is such that the onset of the disease may be sudden or slowly progressive. Consequently, except for a fortuitously obvious etiologic factor, no reliable distinguishing features should be expected clinically.

3. The clinical manifestations of mesenteric vascular disease may be divided into three groups: those which result from spasm or small, non-fatal occlusions of the mesenteric arteries, so-called "abdominal angina" or "abdominal intermittent claudications"; those which follow an extensive occlusion of either a mesenteric artery or vein, "mesenteric vascular occlusion"; and those which are a consequence of the rupture of a sclerotic artery with intraperitoneal hemorrhage, "intra-abdominal apoplexy."

4. The clinical characteristics of each of these three groups have been discussed in detail and certain points in the surgical management of these cases have been outlined.

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# INTERPOSITION OF VITALLIUM PLATES IN ARTHROPLASTIES OF THE KNEE

## PRELIMINARY REPORT

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THE reconstruction of a new joint by arthroplasty is a complicated surgical procedure involving the consideration of many factors. The interposition of fascia lata or other material as a lining of the new joint is only one of several important steps in the operative technique.

The interposition of various metals, as gold and silver, between the articular surfaces of an ankylosed joint is by no means a new procedure, having been tried and abandoned over fifty years ago.

FIG. 1. Case 1. Anteroposterior and lateral roentgenograms of knee following arthroplasty, showing vitallium plate in position.

Smith-Petersen has revived this principle in arthroplasty of the hip joint, employing an entirely different method whereby a non-irritating metal cap is placed over the head of the femur. He first tried various materials for this purpose, as glass and

bakelite, but has recently adopted the use of caps made of the alloy vitallium, which was introduced by Dr. Charles Venable.

Fig. 2. Case 11. Anteroposterior and lateral roentgenograms of knee following arthroplasty, showing vitallium plate in position.

The cap is not fixed in any way to the bone; thus, free play is possible between the cap and the head of the femur, as well as between the cap and the acetabulum. He has employed this method in a small number of cases of bilateral ankylosis as a result of atrophic or rheumatoid arthritis, with excellent results. The procedure was also carried out in one case of ankylosis following a pyogenic infection; sufficient time had not elapsed since this operation to determine the end result.

In one patient, Smith-Petersen demonstrated a fibrocartilaginous lining in the new joint following the interposition of the vitallium cap, similar to the evolutionary physiologic changes observed by the author after the use of fascia lata as a lining membrane, reported in the Robert Jones Lecture in 1930. In Smith-Petersen's cases, however, a longer period of time had elapsed and the evolutionary process had reached a more mature stage at the time of operation than in those of the author; further, a smoother and more normal contour of the interior of the joint was obtained than in the author's cases, wherein fascia lata had been interposed.

Venable has also employed a vitallium cap for lining the joint in arthroplasty of the hip. He fixed the cap by a metal screw, allowing motion only between the cap and acetabulum.

These measures have not been tried by a sufficient number of surgeons, nor has a proper length of time elapsed to permit an estimation of their true value, but the results shown by Smith-Petersen warrant further investigation. With this in mind,

the author has interposed a vitallium plate in two patients with ankylosis of the knee following acute pyogenic infectious arthritis. The technique of the operation is similar to that described by the author for arthroplasty of the knee where in fascia lata is used, although slightly less joint space was created than has been customary for the interposition of the membrane. Prior to the operation, the size of the plate was estimated from roentgenograms and constructed to fit over the anterior surface of the lower end of the affected femur. Since the anatomy of the knee joint precludes the interposition of metal without internal fixation, the plate was maintained in position by two posterior triangular flanges hooked into the posterior surface of the condyles, and by one vitallium screw inserted into the anterior surface of the shaft of the femur.

In the two cases in which this plate has been interposed, we have been disappointed in the amount of motion obtained. This may perhaps be due to an inadequate joint space. In the future, we plan to enlarge the joint space for interposition of vitallium to equal that of joints which are lined with fascia lata. The proper joint space between the articular surfaces of the femur and tibia is approximately ¾ inch.

We are now working on a modification of these plates in order to obtain one which can be more easily removed, if necessary. We are also attempting to model a plate to fit the upper end of the tibia, to be used instead of the plate on the lower end of the femur.

Our results following arthroplasty of the knee wherein fascia lata was interposed have been so satisfactory that this material will by no means be discarded until some other substance can be secured which will minimize the operative procedure and provide an even more normal joint. In science, one should possess an open mind, and if the results can be improved by new methods, they should be adopted. It is in this spirit that we are now using vitallium plates in arthroplasty of the knee. A further report will be made after the trial of this metal in a larger number of cases.*

## CONCLUSIONS

The use of vitallium in the reconstruction of a new joint is in the experimental stage, and the ultimate outcome of these cases is at present doubtful. Smith-Petersen's results in the hip have been encouraging and have led the author to try this method in the knee joint. Sufficient length of time has not elapsed, however, to determine whether or not the interposition of vitallium in the knee will be of permanent value, as the problems connected with arthroplasties in this joint differ materially from those in the hip. A further report will be made on these cases when sufficient time has elapsed to determine the end results.

Acknowledgment is made of the services of Dr. Harold B. Boyd, who furnished material aid in the construction of this cap.

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* Since the submission of this paper for publication, two additional arthroplasties of the knee have been performed wherein vitallium was interposed.

# DISCOGENETIC DISEASE

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I

IN 1824, Wenzel[25] described a disease "in which the intervertebral discs are involved early and almost exclusively, whereby we find at the vertebral bodies a just beginning morbid change which consists in inflammation." About twenty years later, Rokitansky[17] stated that the common type of spondylitis deformans (which is marked by exostoses at the margins of the vertebral bodies) is a sequel of degenerative thinning of the intervertebral discs. Half a century later, and within one year after the discovery of the *x*-rays, Beneke[1] reported the results of his autoptic and roentgenographic studies in an almost inaccessible publication. Confirming Wenzel's and Rokitansky's views on the pathogenesis of spondylitis deformans, he added that static factors are essential in the production of this disease which he grouped with osteoarthritis.

This, as well as Léri's[10] Eugen Fränkel's[5] and Simonds'[21] discerning work on the subject, was almost forgotten when, about twelve years ago, Schmorl and his pupils[19] rediscovered the pathogenetic significance of lesions of the discs in diseases of the vertebral column, especially in the common disease which they termed "spondylosis deformans" (hypertrophic or osteoarthritic spondylitis of the Anglo-American literature). Their observations have since been confirmed by many authors. Experimentally, this type of spondylitis was produced by injury to the discs in dogs,[8] and, by accident, in man.[11]

According to these investigations, the pathogenesis is as follows: Thinning of the discs induces, first, abnormal mobility of the adjacent vertebral bodies, thus causing abnormal stress upon the ligaments that connect them; secondly, abnormal contact between adjacent vertebral surfaces; finally, abnormal position of the articular processes. The stressed ligaments may calcify; contact between vertebral bodies may produce condensation and exostoses; in the displaced facets of the intervertebral (apophyseal) articulations, osteoarthritis may develop; and the diminution of the distance between adjacent vertebrae, together with the displacement of articular processes, may considerably narrow the intervertebral foramina, thus causing pressure upon the nerves that pass through them. Thinning of the discs may be due either to trauma, or to a complex degenerative process, or both; irrespective of its origin, however, it is this thinning which underlies all the changes above mentioned—these changes taking place in vertebral structures that are not primarily diseased. On the other hand, infection or new growth in the vertebrae may secondarily involve the discs; but the mechanical alterations act then upon diseased areas, e.g., softened bone; the results, differing entirely from those observed in normal vertebrae,[13] do not form part of this discussion.

II

Hypertrophic spondylitis may develop within a few months after the discs have been injured;[11,16] but, in other instances, no noticeable changes occur in the vertebrae during indefinite periods of observation. Beneke reported one such case; he believed that, since the patient had been confined to bed for several years, exostoses failed to develop because the normal mechanical stress upon the spine had ceased; and he concluded that persistence of this stress after the discs thinned down is essential in the production of hypertrophic

spondylitis. The following examples may illustrate the fact that other factors also are involved.

A healthy woman incurred a severe trauma of the spine at about 40 years of age. Ten years later, radicular neuritis with wasting of the muscles of one upper extremity had gradually developed and lasted for the subsequent twenty years. About forty years after the accident, when neuritic symptoms recurred, roentgenograms showed obliteration of the fifth and sixth cervical intervertebral spaces, indicative of thinning of their discs, but practically no deformities or condensation of the adjacent vertebrae. Three years later, the aspect of the spine had not changed. (Fig. 1.) Throughout these forty years, however, the patient had led a very active life.

For comparison, Figure 2 shows roentgenograms, taken at an interval of eleven months, of a woman of about the same age and build who also suffered from severe radicular neuritis: the rapid progress of condensation and exostoses during this interval contrasts strongly with the previous instance.

In other cases, some of the vertebrae adjacent to thinned discs form large exostoses, while others remain normal. (Fig. 3.) These observations suggest that persistence of normal stress upon the spine is in itself not sufficient to account for the production of bone hypertrophy; the readiness of the bone to respond to irritation seems to vary in different persons, as well as in different sections of the selfsame vertebral column.

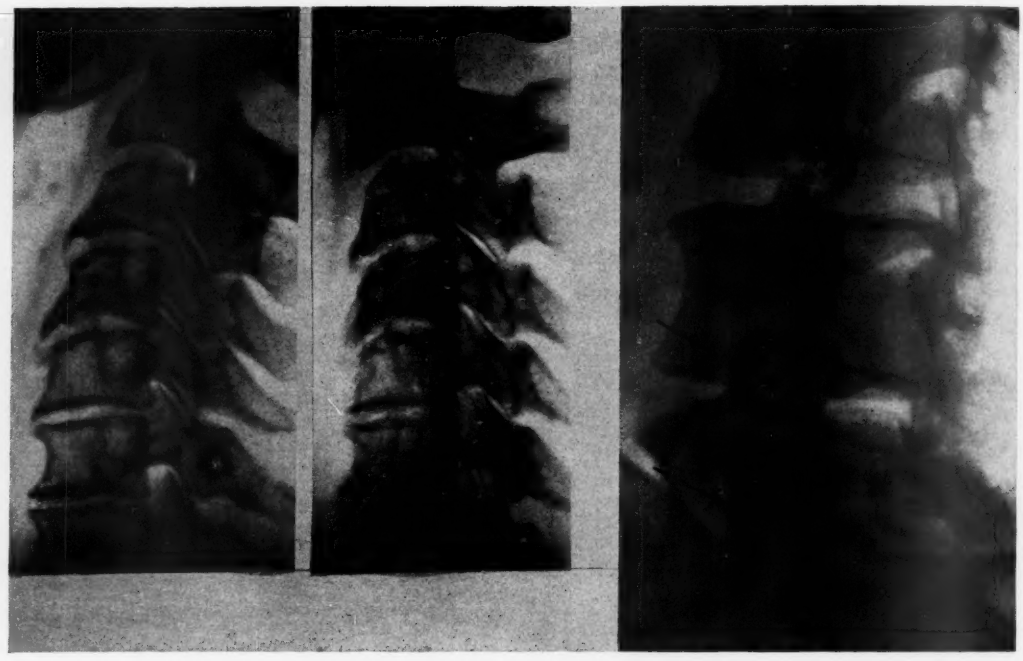
Vertebral exostoses commonly originate at the junction of the annular epiphysis (Schmorl's "Randleiste") with the main part of the vertebral body.[19] This is also the place where the anterior longitudinal ligaments represent to some extent the periosteum of the vertebral bodies.[19] When they are under stress, irritation may lead to "periosteal" ossification as in other bones; this is said to occur when the discs, growing soft, protrude beyond the margins of the vertebrae. Eburnation and exostoses may also develop at the horizontal surfaces of the vertebrae, when these come in contact because the discs thin down. Similar exostoses and ligamentous ossifications, however, may occur when the discs are not diseased, e.g., in infectious spondylitis; and well marked calcification of longitudinal ligaments may, for reasons yet unknown, occur in vertebral sections that are otherwise normal, especially in sections of reduced mobility.[14] The ligaments ossify quite commonly in spondylose rhizomélique (spondylarthritis ankylopoietica; Struempell-Marie disease; falsely termed Bechterew disease in the German literature), chiefly after the intervertebral apophyseal articulations have undergone bony ankylosis.[14] In this disease, usually the ligamenta flava are more severely affected than the other ligaments,[10] but recent reports show that thickening and ossification of the ligamenta flava may also occur as a disease independent of other spinal lesions.[12,14,23]

A B

Fig. 1. A, the fifth and sixth cervical intervertebral spaces are narrowed. No exostoses. Apophyseal joints free. B, three years later, the condition is unchanged. Case referred to in text.

When, as a result of thinning of the discs, the distance between adjacent vertebrae diminishes, their articular processes obviously become displaced. In the cervical spine, where the plane of the facets slopes diagonally backward and downward, the superjacent articular process will slide backward along the subjacent facet; in the

dorsal and lumbar spine, where the plane of the facets is more vertical, the superjacent vertebra slides downward rather than backward. This, together with the greater weight upon the lumbar region, exposes the tips of the articular processes in the lumbar spine to greater stress than in the cervical, when the discs thin down. Osteoarthritis of the intervertebral articulations may develop (hypertrophic spondylarthritis) but it is noteworthy that it fails to do so in the majority of cases.[14]



A B C

Fig. 2. A, cervical spine. Thinning of discs with hypertrophic spondylitis. B, eleven months later. Note progress of exostoses. Apophyseal joints free. C, the lumbar spine of the same patient. The two lower discs are thinned, the articular processes are displaced (arrows), their bony density is increased, and the facets are ragged. However, there are no exostoses at the vertebral bodies. (Hypertrophic spondylitis in the cervical spine, hypertophic spondylarthritis in the lumber region, both due to thinning of discs.)

This behavior is similar to that observed in postural anomalies, e.g., scoliosis, where the joints under stress do not of necessity become diseased. Apparently the variable resistance of the articular cartilage of the apophyseal joints is responsible for these differences. This is a factor distinct from the variable readiness of the bone to form exostoses, for hypertrophic spondylitis and hypertrophic spondylarthritis are not interdependent. Figures 2 and 4 show in conjunction with thinning of discs marked hypertrophic spondylitis with normal apophyseal articulations in the cervical region, but definite hypertrophic spondylarthritis with normal vertebral bodies in the lumbar spine.

Like hypertrophic spondylitis, hypertrophic spondylarthritis occurs also in the absence of any changes suggesting abnormal stress, e.g., in persons otherwise healthy who are infected with entameba histolytica.[14]

It would seem, then, that in thinning of the intervertebral discs, the vertebral bodies, articular processes, and intervertebral ligaments, while invariably exposed to abnormal mechanical stress, do not of necessity develop hypertrophic spondylitis and hypertrophic spondylarthritis; whereas, on the other hand, hypertrophic spondylitis and spondylarthritis may and do occur in the presence of normal discs. This suggests that spondylitis and spondylarthritis of this type are merely responses to various forms of persistent

irritation, either from without (mechanical), or from within (infectious, toxic). Hence, thinning of discs, being only one of the causative factors involved, should not be considered identical with the vertebral morbid changes that it may or may not induce.

III

Broadly speaking, the discs may thin down in any region of the column, for example, when they are affected by injury, infection, or new growth. But in the more common gradual thinning which is called degenerative, two typical localizations are observed. First, in a rare form of kyphosis, the anterior halves of the discs in the midthoracic spine are flattened by the increasing pressure of the vertebrae upon them. Anatomically, this condition corresponds to Schmorl's "Alterskyphose," and, to some extent, to Knaggs' "spondylosis muscularis," possibly also to the one case autopsied by Bechterew. Junghanns pointed out that this type of kyphosis could not develop unless the vertebral bodies of the affected region were comparatively normal, and that softening of vertebrae may be associated with thinning of the discs in some instances. Previous studies gave me the impression that the type of kyphosis here discussed is not a distinct entity, but a more or less accidental occurrence; for in the pathology of the spine much more depends on the reciprocal hardness and elasticity of discs and vertebral bodies than on the absolute resistance of these parts.

Secondly, in the vast majority of cases, the discs become thinned in the lower cervical and lower lumbar regions. (Figs. 2 and 4.) These regions, being the areas of greatest mobility, are supposedly exposed to the greatest mechanical stress under physiologic conditions.

This does not agree, however, with the fact that when the vertebrae are softened, they usually flatten or collapse in the midthoracic spine and, less commonly, in the upper lumbar region, while they remain normal in height at the cervicodorsal and lumbosacral junctions (where the discs are thinned so commonly). This is illustrated

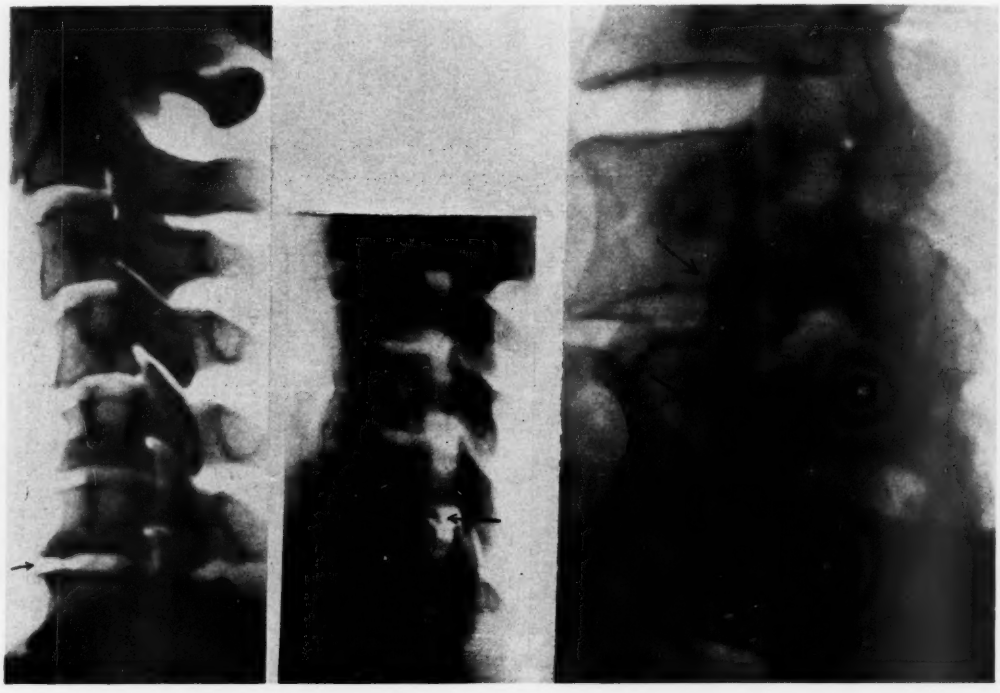
Fig. 3. Hypertrophic spondylitis in thinning of discs. All the lumbar discs are thinned down (*x*), but not all the vertebrae form exostoses (arrows). Apophyseal joints perfectly normal. (Spondylitis without spondylarthritis.)

in Schmorl-Junghanns' book, in which most of the roentgenograms and specimens demonstrate that the midthoracic vertebrae are flattened whenever there is systemic weakening of vertebral bodies. Similarly, in eleven cases of non-localized injury to the spine (fall from a height of more than 6 feet), we found only the midthoracic vertebrae fractured and com-

pressed. This corresponds to the observation that in multiple vertebral fractures the midthoracic vertebrae are those that

IV

As a rule, the clinical symptoms produced by "chronic degenerative" thinning



A B C

FIG. 4. A and B, typical localization of "degenerative" thinning of discs in the cervical spine. C, same in lower lumbar spine. Hypertrophic spondylitis in the certical spine, with exostoses at the posterior borders of the vertebral bodies, bulging into one intervertebral foramen (arrows). Hypertrophic spondylarthritis in the lumbar spine, with displacement of articular processes and narrowing of the apophyseal joint spaces (arrows), in the presence of normal vertebral bodies. See text.

are most commonly involved.[2] In two of our cases, one disc in the lower cervical spine was injured simultaneously with midthoracic vertebrae. The midthoracic vertebrae are also found collapsed in a peculiar type of senile osteoporosis, where the condition may actually amount to a reversed picture of that usually encountered in thinning of discs.[13]

It is difficult to see why the physiologic mechanical stress should be most marked in the midthoracic spine when the vertebrae grow soft, but most pronounced in the lower cervical and lumbosacral regions when the discs lose their elasticity. It seems, however, as though softened vertebrae tend to collapse in the region of physiologic kyphosis, whereas, other things being equal, the discs tend to thin down in the regions of natural lordosis.

of intervertebral discs are quite distinctive. Owing to the common localization in the lower cervical and lower lumbar regions, where the nerves that pass through the intervertebral foramina are especially thick, radicular neuritis is a frequent manifestation. Since the displacement of vertebral structures which produces narrowing of these foramina naturally precedes the formation of any reactive changes in vertebral bodies, ligaments and, especially, in the apophyseal articulations, irradiating pain in the extremities usually precedes the onset of local pain in the back by many years. This "referred" pain is not easily recognized as being of nervous origin, because objective signs of radicular neuritis do not, as a rule, develop at early stages of root compression. Clinically, the neurogenous nature of this "rheumatism" does not

often become evident until wasting of muscles or trophic disorders prove it.[16] Local pain is more common in the lumbo-

Fig. 4D. Thoracic spine normal.

sacral than in the cervical region; it is usually entirely absent in the latter. This agrees with the observation above reported, that hypertrophic spondylarthritis as a result of thinning of discs is more common in the lumbar than in the cervical section.

The clinical course of the disease is usually marked by three conspicuous features. First, the symptoms are not persistent, but recurrent; secondly, they vary in character at different periods; thirdly, they are not proportional to the degree and amount of anatomic changes as found roentgenographically.

Fig. 5. Exostoses at the posterior borders of the vertebral bodies bulge into the intervertebral foramina (arrows). Apophyseal joints free. The case illustrates that minute changes, which escape recognition in conventional roentgenograms, may by their localization give rise to compression of nerve roots.

The clinical signs and symptoms have been described in previous reports.[24] It may here be mentioned that among the 261 patients observed, the majority sought medical advice at about 50 years of age, although the history usually reached back to earlier periods, often even to adolescence. "Rheumatism" or "sciatica" of a few days' duration recurred at intervals of from eight to twenty months. After a number of years the trouble increased in severity, duration, or frequency, until it sometimes amounted to disability of the affected limb. This stage usually lasted for two or three years, after which time the symptoms tended to subside or disappear, although acute exacerbations occurred occasionally. One may finally find the intervertebral spaces entirely obliterated or, in other cases, the vertebrae adjacent to narrowed interspaces fixed by fusion of contiguous spondylitic exostoses; in either case, there are often no more symptoms.

As in other types of spondylitis, riding a car or taking a long walk often brings on the pain; but it is almost pathognomonic of thinning of discs that the pain does not subside during rest at night (as it does in inflammatory diseases of the vertebrae and intervertebral joints); on the contrary, pain after midnight is a very common complaint.

According to these observations, it would seem that weakened discs thin down at intervals, possibly after slight traumas such as may be incurred in daily life. Schmorl found in degenerating discs small fissures at their margins and attributed these lesions to repeated slight traumata. He considered them responsible for leakage of disc material. Correspondingly, one may find under fortunate circumstances that the intervertebral spaces remain of constant width for a period of years, but diminish rather suddenly at irregular intervals. (Fig. 3.) By the narrowing of the intervertebral foramina thus produced, as well as by leakage of the gelatinous disc material backward, compression of nerves may then occur where they are thick. Since the leaking material gradually dries out, the original amount of compression diminishes after a certain period, until a new fissure brings on another recurrence. Since the lordotic position narrows the foramina,[3] movements and positions associated with lordosis commonly accentuate the discomfort. In the lumbar spine this results from backward stretching, and in the cervical by the supine position during rest. When actual herniation of the discs occurs into the spinal canal, the compression becomes permanent in many cases.

The treatment consists in the prevention of any injury to the spine. Avoiding sudden movements, wearing rubber soles and heels, supporting the neck at night by the addition of pillows, are often very helpful measures. In acute exacerbations, ultrashort wave therapy is the most effective treatment known at present. In radicular neuritis associated with trophic disorders of the skin, muscles or bones, one is impressed with the rapid recovery under this treatment of tissues that seemed to be very seriously damaged.[16] In other less dramatic cases a second recurrence often fails to respond to this treatment as favorably as the first.

Irritation rather than relief may be caused by too frequent or too strong short wave applications. Three treatments a week, of fifteen minutes each, given during a fortnight, are usually sufficient. The patient should not experience any sensation during the treatment.

We have now given up manipulations and massage, since they often tend to increase pain. Exceptionally, when short wave treatment is not successful, Roentgen therapy over the diseased area may prove to be helpful (100 r, 155 kV, 4 ma., 0.5 mm. Cu; at intervals of about one week).

V

It has often been observed that compression fractures of vertebral bodies may remain unnoticed by the patient; and it is well known that destruction of vertebral bodies, e.g., in Pott's disease, may cause no symptoms for a long time. According to a recent report,[6] tuberculosis of the intervertebral discs causes severe radicular neuritis. While large exostoses at the anterior borders of the vertebrae are often accidentally discovered in the absence of symptoms referred to the back or limbs, small exostoses at the posterior borders may be responsible for severe segmental neuritis[16] when they bulge into the foramina. (Figs. 4 and 5.)

These findings may throw some light on the much debated question of the actual clinical significance of hypertrophic spondylitic changes. Neither the presence nor the absence, nor the amount of bone changes determines the severity of the clinical symptoms. The degree of thinning of the discs is by no means proportional to the discomfort experienced by the patient. Both the vertebral bodies and the articular processes may become immobilized by fusion of exostoses, in which case pain

ceases although the bone changes are very pronounced. The clinical syndrome associated with spondylitis and spondylarthritis is no more dependent on the amount of "spiculations" or deformities than, for example, the clinical course of pulmonary tuberculosis depends on the amount of conspicuous shadows found in the roentgenogram of the chest.

By the addition in recent years of a number of new facts to the pathology of the spinal column, our conception of spinal disease is being altered at present, and the classifications in use are no longer adequate. The large number of terms and the lack of conformity in their interpretation seem to indicate that these classifications have ceased to be entirely satisfactory. For instance, the disease here discussed is variously termed spondylosis muscularis, spondylitis or spondylosis deformans, trophostatic osteoarthrosis, hypertrophic spondylitis, polyspondylitis marginalis osteophytica, arthritis of the spine, and Bechterew's disease—the two latter terms being also used to designate spondylarthritis ankylopoietica, a condition which has almost nothing in common with the former.

It does not seem justifiable to add another name to this confusing variety, unless this name serves to define a distinct entity and to eliminate terms the meaning of which has become incorrect, dubious, or merely dogmatic. Since in the disease here under discussion a group of anatomic alterations in vertebral bodies, intervertebral articulations, and ligaments, as well as a distinct though variable clinical syndrome, originate in a disease primarily affecting the discs, the term "discogenetic disease," suggested in previous reports, is here adopted as a tentative abbrevation. It may serve to define a common clinical and anatomic syndrome and to distinguish it from the type of spondylitis and spondylarthritis with which it may or may not be associated, but with which it is not identical. To characterize these vertebral changes more fully, one may speak of the discogenetic form of spondylitis and spondylarthritis.

## SUMMARY

There is no constant interrelation between thinning of the intervertebral discs and hypertrophic spondylitis and spondylarthritis. The production of clinical symptoms depends neither on these bony changes nor on the amount of thinning of the discs, but on the localization of the entire process and the stage of the disease. In degenerative thinning, the lower cervical and lower lumbar regions are most commonly involved, wherefore, under certain conditions discussed in some detail, a group of clinical symptoms occurs frequently as a result of this affection. The clinical course, roentgenologic aspect, and mode of treatment are described. Since spondylitis and spondylarthritis are neither characteristic of this disease nor responsible for all the clinical symptoms, it is suggested that this group of anatomic findings and clinical manifestations be designated by the term "discogenetic."

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Fracture of the publc portion of the pelvic ring . . . is the most common form of fracture occurring in the pelvis, and is caused usually by a squeeze between two solid objects or a fall from a height, the patient striking on the hip. It is quite frequently associated with other fractures in the pelvis.

From—"Fractures" by Paul B. Magnuson, 3rd Edition (Lippincott).

# CASE REPORTS

## SURGICAL MANAGEMENT IN CASES OF ADVANCED DISEASE OF THE BILIARY TRACT

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ONE in every ten adults has gallstones and at least one more has cholecystitis without stones. They form a vast army of potential candidates for the complications of disease of the biliary tract. These complications are so numerous and so easily acquired that the candidate is sure of election if he lives long enough. With this thought in mind, the physician who undertakes the management of these patients by non-surgical measures assumes a grave responsibility. Medical treatment undoubtedly will be often successful in the control of symptoms and thereby it will confer a false sense of security upon both patient and physician. In the column of the surgical dyspepsias, disease of the gall-bladder leads all the rest.

The symptoms of cholecystitis are the most irregular and capricious of any lesion within the abdomen. But, once the physician has had his suspicions aroused he should not be content until the gall-bladder has been convicted with reasonable certainty, or unequivocally cleared of all suspicion. One must not be misled by the cholecystogram. When it indicates disease of the gall-bladder by definite signs such as non-visualization or filling defects due to stones—then it is accurate in over 90 per cent of cases. However in similar cases, clinical diagnosis, after careful interpretation of the history and physical examination, is also accurate in 90 per cent of cases. A gall-bladder which visualizes and empties in the cholecystogram may be markedly diseased and even contain stones. In this connection it is well to remember three facts:

1. Filling of the gall-bladder with bile depends on a functioning sphincter of Oddi at the lower end of the common duct.

2. The diseased gall-bladder, except in certain rare, usually acute conditions, can and does concentrate bile as witness the thick viscid bile in the diseased gall-bladder and the usually intact often hyperplastic mucosa and submucosa.

3. The wall of the diseased gall-bladder commonly is much thickened, a condition which the microscope reveals to be caused by marked hypertrophy of the intrinsic muscle fibers. This certainly indicates that the contractile and emptying mechanism has been overworked, probably because of some impediment to the outflow of bile.

There is an increasing tendency today to temporize with gall-bladder disease by so-called gall-bladder drainage, by diet, etc. Some of this is due to the fact that the physician hesitates to advise operation until urgent complications appear; or because he has had the unpleasant experience of mortality or morbidity. It goes without saying that surgery of the biliary tract is not the work for the inexperienced though daring tyro. In proper hands the risk is a reasonable one—no greater than that of any other major operation. It is not fully appreciated that cholecystitis, when once established, is a going concern that may have exacerbations and remissions but always produces a steadily progressive destruction. Is it not a fact borne out by

operating room experience that in every patient with disease of the gall-bladder who comes to operation there is visible damage to the liver; inflammatory changes in the common duct (which I was able to show by microscopic examination); and too often a swollen pancreas due to lymphangitis or to interstitial changes? Although serious enough, these are mild complications compared with others which may follow procrastination and delay.

Mrs. A. R., aged 62 years, was admitted to St. Joseph's Hospital April 5, 1937 with the chief complaint of jaundice which had been present for two weeks. About eight weeks before admission she had an attack of biliary colic which was relieved by a hypodermic of morphine. She recovered completely, but about four weeks later she had another attack of pain, not as severe as the first, followed in about forty-eight hours by the appearance of jaundice which became more marked and persistent until her admission to the hospital. Her only other complaint was a dull burning feeling in the right upper abdomen.

She had suffered from attacks of indigestion for a number of years but never had severe pain. Otherwise her general health had always been good. Her usual weight was 250 pounds.

Physical examination revealed an obese female 5 feet 6 inches tall weighing 210 pounds. She had no pain or discomfort. There was a generalized jaundice. Examination of her heart and lungs revealed nothing abnormal. Her blood pressure was 110/60. The abdomen was fat, soft and presented no palpable masses and no area of tenderness. In other words except for the presence of jaundice the patient did not appear to be ill. Her appetite and sleep were normal.

During the following two weeks there was very little change in her condition except several slight episodes of epigastric pain with radiation to the right shoulder and the fact that the jaundice persisted.

*Laboratory Tests.* The urine showed a faint trace of albumin, a few granular casts and the presence of bile. The blood count showed a slight leucocytosis. The blood Wassermann was negative. The Van Denberg test made on a number of occasions showed an immediate direct reaction and the icterus index varied between a low of 110 on May 3, 1937 and a high of 170 on April 21. The blood sugar was 87 and the blood urea nitrogen 15.

I first saw the patient sixteen days after her admission, at which time I believed she had a stone in the common duct, although, according to the color of the stools, a small amount of bile had been coming through for two days. According to the temperature chart there was no infection. I recommended taking another icterus index, with operation if this showed the same or a higher value.

The stools became normal in color, indicating that the obstruction was somewhat relieved and there was improvement in the patient's general well being. She had a good appetite, no abdominal pain or tenderness, and normal temperature. She was therefore allowed to go home on May 4, 1937 under the care of her family physician.

The *x*-ray findings were interesting. The day after admission a flat *x*-ray plate of the abdomen indicated that the gall-bladder, filled with milk of calcium, was slightly enlarged. Two days later the patient was given the Graham dye and the *x*-ray revealed exactly the same density in the region of the gall-bladder previously reported. There was no evidence of non-opaque stones.

The patient was readmitted May 24th, with jaundice still present but somewhat decreased. The icterus index at this time was 45. The blood count showed a mild secondary anemia.

Operation was advised and carried out under ether anesthesia on May 26, 1937. An upper right rectus incision was made and the stomach and duodenum were found normal. The liver was enlarged, of slaty color and with rounded edges. The head of the pancreas was uniformly enlarged but not indurated. The gall-bladder was pale, distended and filled with stones. The common duct was dilated to a diameter of 2 cm. and several stones could be palpated floating up and down in the duct. When it was opened, a gush of dark bile appeared and two stones were found each 1 cm. in diameter, round and non-faceted. A curved hemostat was passed down the duct and into the duodenum, and the common duct was again palpated throughout. No more stones could be discovered. A No. 20 French rubber catheter was sutured in place in the common duct with its end upward toward the liver. The opening in the duct closed around it. The gall-bladder was then removed in the usual manner, from below upward, and the liver fossa oversewed

with catgut. There was very little bleeding at any time and the field was dry at the end of the operation. Two cigarette drains were placed in the subhepatic fossa, below but not in contact with the common duct. The wound was closed in layers around this drainage. The patient was given 500 c.c. of blood intravenously by the citrate method at the conclusion of the operation and she left the table in good condition.

The postoperative course was unusually smooth. Bile drained freely from the tube in the common duct. The two cigarette drains were removed five days after the operation and the catheter in the common duct came away spontaneously on the fifteenth day. The wound healed by primary union. The stools were of normal color and the jaundice was almost cleared at the time the patient was discharged from the hospital on the twenty-seventh day.

I saw this patient on July 28, 1937 at her home. She was feeling entirely well, had been enjoying walks out-of-doors and had no complaints referable to her old trouble.

From the standpoint of diagnosis this patient's history is rather typical of stone in the common duct, that is, attacks of indigestion over a period of years with recent biliary colic followed by jaundice. The only missing symptom was fever, which of course is indicative of cholangitis and is nearly always present in cases of this nature. Its absence in this patient did not necessarily mean absence of infection along the common duct or in the smaller ducts within the liver. I believe that in all such conditions definite evidence of infection can be found, although it may not be present in sufficient degree to give rise to systemic manifestations.

It should not be forgotten that biliary colic followed by jaundice may be due to obstruction at the lower end of the common duct either by pancreatitis or by carcinoma of the head of the pancreas. In other words, pain ess obstructive jaundice is usually caused by pancreatic malignancy, but on the other hand malignancy of the pancreas may occur in a patient who has gallstones. The patient with obstructive jaundice, regardless of whether the symptoms point to stone or cancer, nevertheless should be given the benefit of exploratory operation. It is not always possible to be certain of the diagnosis even when the pancreas is in the hand. I have seen a number of patients who had been operated on by experienced surgeons where the diagnosis of carcinoma of the head of the pancreas was proved to be erroneous by subsequent events.

When should operation be done in the patient with obstructive jaundice? How long is it safe to wait? What are the dangers and how can they be minimized? These are practical questions. The answers will often determine success or failure. Conditions vary in the individual patient so that it is difficult to lay down rigid rules of procedure. Much depends upon surgical judgment.

In general it may be stated that the patient with jaundice does not present an emergency problem. There is time for detailed examination particularly to note whether the jaundice is increasing or decreasing. This can be accurately determined by the icterus index. From a practical standpoint the color of the stools is important because it indicates whether or not bile is entering the intestine. When there is evidence that jaundice is decreasing and bile flow is becoming reëstablished it is advisable to postpone operation because in the presence of complete biliary obstruction there is a profound disturbance of the function of the liver which makes any surgical procedure extremely hazardous. A waiting policy does no harm unless there is active infection of the cholangitis type. In this event, and in spite of increased risk, external drainage of bile should be established by operation. However, this must be carried out as a minimum procedure; nothing should be done of a corrective nature such as removal of the gall-bladder or investigation of any other lesion. These should be left to a later time when the patient has passed the acute crisis.

In all these conditions the surgeon's chief ally is the wonderful recuperative power of the liver. There are on record cases of complete biliary obstruction of several

months' duration which have been relieved by the proper surgical procedure. I saw a patient not long ago who had complete biliary obstruction for eighteen months. He was deeply jaundiced, the liver was markedly enlarged but he was up and about and feeling fairly well. Operation was not done and at autopsy the obstruction was found to be due to stones in the common duct.

The chief dangers of operation in these patients are failure of liver function, renal insufficiency and hemorrhage. These can be minimized to a large extent by special preoperative preparation which should include blood transfusion, high carbohydrate intake and calcium chloride and glucose intravenously. The tendency to bleed in these patients presents a difficult problem. Frequently it is the cause of death in spite of the most careful safeguards. Notwithstanding the guidance of laboratory tests and the use of preventive measures, we know from surgical experience that some patients have uncontrollable bleeding and there is no certain way by which this can be foretold or prevented.

*Operative Technique.* I use an upper right rectus incision of adequate size beginning at the xyphoid notch. The suspensory ligament of the liver is divided to aid exposure. Palpation of the common duct with a finger in the foramen of Winslow, examination of the gall-bladder and investigation of the head of the pancreas are carried out. The stomach, duodenum and hepatic flexure of the colon are held firmly to the medial side by gauze pads under the hand of an assistant whose fingers straddle the gastrohepatic ligament. A clamp on the fundus of the gall-bladder enables one to pull it down and out and this makes accessible the extrahepatic structures. The common duct is opened by a small longitudinal incision and is then explored by a probe or curved hemostat for free passage into the duodenum. Usually these maneuvers will dislodge stones and milk them up toward the opening in the duct, from which they can be removed. A rubber catheter with a small eye cut in its side is placed in the duct with its end upward toward the liver. It is fastened in place by one suture and then the opening in the duct is closed around it.

Attention is now directed to the gall-bladder which in most cases I prefer to remove. First, the cystic artery and cystic duct are clamped in one bite of the hemostat after the neck of the gall-bladder is separated from its loose attachment to the liver. I always remove the gall-bladder from below upward and try to do this mostly by sharp dissection while clamping any visible vessels which appear along the liver attachment. Before the gall-bladder is completely removed the pedicle containing the cystic artery and cystic duct is doubly ligated with chromic catgut. The gall-bladder bed is oversewed with catgut in order to cover raw surface and to control oozing. A dry field is desirable but not always possible and in such instances I do not hesitate to place a narrow double strip of iodoform gauze against any oozing raw surface of the liver. A cigarette drain is placed in the subhepatic fossa in order to guide any leakage of bile to the outside. The wound is closed in layers around the drainage material. The end of the catheter is placed in a 6 ounce bottle which is fastened to the dressings.

The complications of disease of the biliary tract present some of the most bizarre features encountered anywhere in pathology. The history of this patient is of unusual interest.

A woman, 69 years of age, was admitted to the hospital December 2, 1936 complaining chiefly of abdominal pain associated with distention and absence of any bowel movement for four days. The onset was sudden following a dose of sal-hepatica. This was followed by colicky pains on the left side of the abdomen which persisted to the time of her entrance to the hospital.

The chief features on physical examination were distention of the abdomen, exaggerated peristaltic activity, but no areas of rigidity or tenderness. The blood count revealed a leucocytosis of 14,200. The Kahn test was negative.

The urea nitrogen was 13, the blood sugar 91. Examination of the urine revealed nothing abnormal.

When the patient was admitted to the hospital she was seen by another surgeon and it was his opinion that she was suffering from intestinal obstruction. A barium enema, on the day of admission, revealed no evidence of obstruction in the colon, sigmoid or rectum. Two days after her admission a sigmoidoscopic examination was made without revealing any evidence of obstruction, but nevertheless, because of the patient's history and physical findings there was still a strong suspicion that she probably had a malignant obstruction somewhere in the colon. During this time the patient's condition improved somewhat as a result of repeated enemas and the distention almost entirely disappeared. Her temperature, pulse rate and respirations were normal. She still complained of griping abdominal pain.

I saw the patient five days after her admission, at which time examination of the abdomen could be done with a fair degree of satisfaction. The chief feature of this examination was a palpable, very hard round mass below the right costal margin which was slightly movable, not tender and apparently attached to the liver. I suspected that this was probably a metastatic carcinoma of the liver, but in view of the uncertainty of the diagnosis and the clinical evidence of subsiding intestinal obstruction, it seemed advisable that operation should be done.

An upper right rectus incision was made. The gall-bladder was surrounded by dense adhesions which fixed it firmly to the hepatic flexure of the colon. Separation of these adhesions revealed a fistulous communication between the gall-bladder and the transverse colon; the opening in the colon was about the size of a fifty-cent piece. The edges of this opening were trimmed down to healthy tissue and closure made by two rows of catgut suture reinforced by interrupted linen sutures. The markedly thickened walls of the gall-bladder were contracted around one large faceted stone. Because of the inflammatory edema in the region of the cystic and common ducts, the gall-bladder was freed from above downward and then resected, leaving a fairly good sized stump which was oversewed to control bleeding. The gall-bladder bed in the liver was oversewed. A double strip of iodoform gauze was placed against the liver bed, surrounded by three cigarette drains and the omentum interposed between these drains and the transverse colon. The wound was closed in layers around the drains.

The immediate convalescence was uneventful. About ten days after the operation the patient began to complain of severe abdominal cramps which occurred almost every day and on several occasions were associated with nausea and vomiting. These attacks closely resembled intestinal obstruction but they always relieved themselves spontaneously. Because of their severity and tendency to recur I had almost decided to reopen the abdomen. At this time a rectal examination was made and I could feel a hard mass which was movable and seemed to be located in the region of the sigmoid. Another rectal examination two days later revealed a large gallstone in the rectum which was removed after gentle dilatation of the anal sphincter. This was followed by a complete disappearance of the patient's symptoms and within three days she was discharged from the hospital feeling entirely well and with the abdominal wound completely healed. I saw the patient at intervals during the following year. She remained in good health and had no abdominal complaints.

In retrospect, we can reconstruct the events which took place in this patient. She had, to begin with, two large stones in the gall-bladder; the gall-bladder formed an inflammatory attachment to the hepatic flexure of the colon with which it established a fistulous connection; through this fistula one of the stones passed into the hepatic flexure of the colon and because of its size it gave rise to the attack of intestinal obstruction which brought the patient to the hospital. The stone, through repeated attacks of obstruction, finally found its way to the rectum from which it was removed.

Twelve years ago, with the late Dr. E. S. Judd, I reported 153 cases of internal biliary fistula from the Mayo Clinic, the largest single series on record. In preparing this report we had occasion to review the literature and it was astounding to learn the bizarre types of biliary fistula which had

been recorded. For instance, gallstones have been vomited from the stomach; they have been coughed up from the bronchi; they have been voided with the urine and have been found impacted in the male urethra and also removed from the bladder by lithotomy; they have been found in the pregnant uterus, in the contents of an ovarian cyst; they have been found in the pericardial and pleural cavities; and the autopsy performed on Ignatius Loyola revealed that his portal vein contained a number of gallstones. From the review of these cases and a study of the literature the following facts were deduced: (1) The fistula does not cure the disease; (2) it rarely occurs in the absence of stones; (3) the organs involved in their order of frequency are the duodenum, the colon and the stomach; (4) the fistulous connection usually is by direct communication—in other words, rarely is there an intervening abscess cavity; (5) intestinal obstruction is very uncommon in these cases; (6) the diagnosis is made only at the operating table; (7) the mortality is 10 per cent.

Whenever the gall-bladder is adherent to neighboring structures, separation should be carefully done and the freed organs always should be examined for a fistula which may be small and escape detection. It is advisable to remove the gall-bladder whenever conditions permit. The common duct should always be examined for the presence of stones and, of course, when they are found they should be removed.

In recent surgical meetings the subject of the management of acute cholecystitis has come in for considerable discussion. There are those, and they have the backing of the older element of surgical authority, who advocate withholding operative interference until the acute clinical signs have subsided. There are strong arguments, chiefly those of extended experience, to support their opinion. On the other side are the advocates of immediate operation for reasons analogous to factors in acute appendicitis.

I feel that much of this controversy is due to misunderstanding. If an experienced surgeon from each group were to go to the bedside of an individual patient suffering from acute cholecystitis I believe that there would not be much difference of opinion as to the surgical management. It must be understood that the ordinary acute biliary colic does not constitute acute cholecystitis. No surgeon regards this condition as a surgical emergency.

My understanding of acute cholecystitis from the clinical standpoint is as follows: After a period of upper abdominal distress characterized by bloating, impairment of appetite, nausea and vomiting, there is the onset of pain, not very acute, in the right upper abdomen associated with moderate local tenderness and soreness which does not subside in reasonable time. Associated with these symptoms is the general appearance of illness, with a marked febrile reaction, although no chills, and possibly jaundice, after forty-eight or seventy-two hours. The apparent severity of the patient's illness far surpasses the physical findings elicited by abdominal examination.

In a patient of this description I believe that wise surgical judgment is best served by masterful inactivity coupled with close daily observation to detect the sudden appearance of acute complications. If we could look inside the abdomen of this patient what would we see? The gall-bladder would be acutely inflamed, partially distended and surrounded by fresh inflammatory adhesions, bathed in free peritoneal fluid. The liver would be congested and its enveloping peritoneum would be inflamed, particularly in the area adjacent to the gall-bladder. The common duct and cystic duct would be difficult to identify because of inflammatory changes in the surrounding tissue and the glands along the extrahepatic biliary tract would be enlarged. This lymphadenitis and lymphangitis probably would have extended down to and around the head of the pancreas. If the microscopic eye could explore the interior of the liver it would reveal that the acute

inflammation even had extended along the course of the small bile channels within the liver lobules.

Would it be wise in these circumstances to invade this hotbed of unrest in which the forces of nature are waging a successful battle? Is one justified in introducing what from a biologic standpoint is a crude and clumsy procedure, namely, removal or even drainage of the gall-bladder? Can this intervention serve any useful purpose when it is realized that the infection with its inflammatory reaction is not in a subsided or even subsiding stage and when its main focus is by no means localized or confined to the gall-bladder?

One of the arguments advanced for immediate operation in cases of acute cholecystitis is the potential danger of rupture or perforation of the gall-bladder. This is a very uncommon condition and usually when it does occur the surgeon has not had the opportunity to see the patient at the onset of his illness. Rupture of the gall-bladder may, of course take place while the patient is under observation, but when it does it is usually evidence of neglectful observation. Every patient with acute cholecystitis who is under observation should have at least a daily examination of his abdomen with particular attention to the development of acute physical signs. At times the development of disease in the gall-bladder becomes fulminatingly acute in a very short time. This is exemplified in the following report:

One Sunday night a man 60 years of age was referred to my service in the hospital because of acute abdominal pain. He stated that two weeks previously he had an attack of abdominal pain which was not severe and lasted about two hours. On the day before admission he was seized with an attack of severe pain in the right side of the abdomen, followed by vomiting which lasted throughout the day. There was nothing of a pertinent nature in his past medical history and he especially stated that his appetite and digestion had always been of the best.

The chief features of the physical examination were as follows: The abdomen was distended and peristalsis was active. There was an area of localized exquisite tenderness on the right side of the abdomen somewhat toward the flank and about midway between the costal margin and the umbilicus. This area was very tender to the slightest pressure.

The temperature was 100 and pulse 108. A blood count showed a leucocytosis of 19,250. The urine was normal.

I felt that here was an acute surgical abdomen which demanded immediate operation. Because of the history of rapid onset of acute pain followed by nausea and vomiting and the physical findings, I suspected that we were dealing with acute appendicitis with the appendix occupying a high position.

Operation under spinal anesthesia was carried out a short time after the patient's admission. A mid-right rectus incision was made. Examination of the appendix revealed no gross changes. The omentum was strongly adherent to the parietal peritoneum in the right upper abdomen and could be separated only by sharp dissection. We then saw that we were dealing with a tightly distended thick gall-bladder, the distal part of which was black and gangrenous and in which an area about the size of a fifty-cent piece was of paper thinness, obviously on the verge of impending rupture. The gall-bladder was freed from its surrounding omental adhesions and only then could an examination be made of the region of the cystic duct. This was found markedly thickened by inflammatory and edematous tissue which made it utterly impossible either to identify the important structures in this area or to consider an attempt to remove the gall-bladder. The fundus of the gall-bladder was opened, releasing thick hemorrhagic material and a number of stones of various sizes. One large stone was impacted in the neck of the gall-bladder and was removed only after some difficulty. A split rubber tube of large caliber was placed well down into the gall-bladder and then sutured in place by attaching it with a through and through suture to the opening. The opening was then closed by suture around the tube. Two cigarette drains were placed outside the gall-bladder and the omentum carefully tucked around these. The wound was closed in layers around the drains.

The patient's convalescence was without incident. The tube in the gall-bladder came away spontaneously on the tenth day and the cigarette drains had been removed three days

previously. Bile drained freely from the tube and later from the wound and continued to do so for about twenty days. The patient was discharged from the hospital twenty-five days after the operation with his wound soundly healed and apparently in good general condition. I saw him at my office at intervals for eight months after his discharge from the hospital during which time he remained entirely well.

A married woman, aged 48 years, was admitted to the hospital February 1, 1938 because of attacks of biliary colic associated with jaundice. She had her first attack twelve years before, beginning as a crunching pain in the epigastrium and radiating around the costal margin to the angle of the scapula. It was followed by jaundice which lasted four or five days. The next attack occurred nine years later and was similar to the first attack. During the past three years she had been experiencing attacks of severe biliary colic always followed by jaundice and occurring about every three or four months. The last episode occurred five months before admission and for the first time it was associated with chills and fever and jaundice of one week's duration. In twelve years the patient's condition between attacks had been characterized almost continuously by heartburn and epigastric distress, consisting of fullness and bloating especially after eating greasy and fried foods.

There was nothing of a relevant nature in her past medical history. A cholecystographic study made a month before admission showed the presence of gallstones. The physical examination revealed nothing of importance except tenderness on deep pressure directly over the gall-bladder. The temperature, pulse and respiratory rate were normal. There was no evidence of jaundice. The examination of the urine was normal. The blood count showed a hemoglobin of 13 mg., red blood cells 4,100,000 and white blood cells 14,000.

Operation was carried out ten days after admission under ether anesthesia. An upper right rectus incision was made. The liver presented a nutmeg appearance and the edges were rounded. The gall-bladder was hugely distended and contained many stones. The common duct was dilated to a diameter of 5 cm. and was tightly packed throughout by stones and sand and these extended also up into the main hepatic duct. The common duct was opened through a longitudinal incision and at once thick dark bile with stones of various sizes and sand gushed forth. The common duct and the hepatic duct were emptied of their contents with the aid of scoops and forceps and finally by flushing with normal saline through a rubber catheter. Both ducts could be easily explored with the finger throughout the entire extent so that one could feel fairly sure that no more stones were left behind. The head of the pancreas was not enlarged. A No. 20 French catheter was placed in the common duct with its end pointed upwards in the hepatic duct and fastened in place by a suture of catgut. The opening in the duct was then closed around this catheter and bile began to drain freely from the tube.

The gall-bladder was removed from below upward and the gall-bladder bed oversewed with catgut. The stomach and duodenum were normal. The appendix was atrophic and was not disturbed. The uterus contained a number of small fibroids. A cigarette drain was placed in the subhepatic fossa below but not in contact with the common duct. The wound was closed in layers around the two drains.

The patient's postoperative convalescence was uneventful. At first the bile draining from the catheter was a dark muddy brown with a heavy sediment but later it became a light syrupy golden yellow. The amount of bile drainage varied between 450 and 650 c.c. daily. The color of the stools beginning with the first bowel movement on the fourth day was a normal brown color. The cigarette drain was removed on the eighth day and the tube in the common duct came away spontaneously on the fifteenth day. The patient had two attacks of auricular fibrillation which yielded promptly to digitalis therapy. The patient was discharged on the twenty-third day with the wound completely healed. At the last examination two months after the operation she was entirely well.

The noteworthy features in this report are: (1) the enlarged distended gall-bladder containing stones associated with numerous stones in the common duct; (2) the absence of jaundice for five months preceding operation in spite of the fact that the hepatic and common ducts were tightly packed with stones and sand; (3) the huge distention of the common duct which measured 5 cm. in diameter.

The association of a contracted gall-bladder with stone in the common duct is a surgical maxim that has come down through the years. The necessary time for the development of jaundice in the event of obstruction of the common duct is considerably shorter when the gall-bladder is functionless through disease or absent than when it is present and capable of receiving and concentrating bile. Nearly all stones in the common duct have had their origin in a diseased gall-bladder and this accounts for the advanced cholecystic changes. However, there is ample presumptive evidence of the formation of stones of successive vintages in the common duct itself. Perhaps the first one or two stones formed in the gall-bladder, passed through the cystic duct into the common duct where their presence gave rise to partial or intermittent obstruction with inflammatory changes. So far as we know this combination of circumstances is most conducive to the formation of gallstones.

I believe this to be the explanation of the great number and varied sizes of stones in the common duct of this patient. The hepatic and common ducts were hugely dilated and literally packed with stones which varied in size from about 2 cm. to fine sand. Yet in spite of these many impediments to free passage, the bile succeeded in percolating through this mixture of gravel in sufficient amount to prevent the development of jaundice except after attacks at long intervals.

## SUMMARY

Advanced disease of the biliary tract gives rise to surgical problems in which disturbed physiology, mechanical factors and infection play interdependent rôles. Four surgical cases are reported and discussed:

1. Obstructive jaundice caused by stone in the common duct.
2. Internal biliary fistula.
3. Acute gangrenous cholecystitis with stones.
4. Calculous common duct without jaundice.

# PROGRESSIVE POSTOPERATIVE GANGRENE OF SKIN*

## REPORT OF A CASE

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IN the course of postoperative complications we have observed a hitherto rare disease—a slowly progressive gangrene of the abdominal wall. According to reports in the literature, Cullen was the first to observe the condition, after the removal of a suppurated appendix, in 1924. The same year Christopher reported a similar process following rib resection for empyema thoracis. In 1926, Brewer and Meleney, Mayeda, Diebold, and Stewart-Wallace dealt with the question. Küppers, Doricev, Coacley and Klein, Branberg, Andersen, Liedberg, Cox, Blaxland, Willard, Wachs and Guszman contributed to the clearing up of this disease with case reports and, in 1935, according to Stewart-Wallace, thirty-seven cases had been reported.

At this writing, the number of reported cases is about sixty, (1924–1939) which is rather low. Probably some of the fatal cases have not been published.

As new evidence shows that we are dealing with a clinical entity, it would be advisable to keep the nomenclature uniform in order that all cases may be followed up. Brewer and Meleney were the first to call the entity "progressive postoperative gangrene of the skin," which seems suitable as it expresses the essence of the process.

This lesion usually occurs in the male (about 80 per cent), especially in the adult. The preceding operations were, in one-half of the reported cases, due to suppurated appendices, in 30 per cent to empyema thoracis, in 15 per cent to suppuration of the upper abdominal cavity, gastric perforation and subphrenic abscess and in the remaining 5 per cent to other operations, mainly abdominal. It was reported in but three instances after aseptic abdominal operations.

The process usually begins within a week following the operation, and occasionally during the second week. One observation was made twenty-one days after operation. Often the fever was gone, the pulse normal and the patient well; in the case reported by Patterson, the patient had been discharged from the hospital.

### CASE REPORT

A male, aged 45 years, was admitted to the clinic on October 28, 1937. Months before, the patient had complained of severe pains in the abdomen and for one month of loss of weight due to polyarthritis. Two days before admission he had severe abdominal pain which soon localized in the lower right side of the abdomen. He suffered from nausea, vomiting, fever and chills, and had lost much weight.

There was marked muscular rigidity of the abdomen especially around McBurney's point and there was an apple-sized, hard, painful resistance at this point. The Rovsing symptom was positive. The white blood cell count was 17,000, the temperature 38°C., the pulse 110. The tongue was coated and furred. Immediate operation was deemed necessary.

A right McBurney incision was made. Behind the cecum a fetid abscess, the size of a fist, was opened and drained with gauze. The abdominal wall was closed with interrupted sutures about the gauze drains.

For a week following operation, the patient was very ill, hiccoughing and vomiting. His abdomen was greatly distended, but enemas were ineffectual. The wound showed a profuse drainage of pus. At the end of the week the hiccoughing stopped and gas was passed. Nineteen days after operation the wound showed an area of edema, the edges became separated and were reddened and painful. The redness spread toward the healthy parts of

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the skin and necrosis was observed in the wound edges. Twenty-three days after operation, the subcutaneous connective tissue of the wound was granulating well, but the necrosis of the skin margins already extended 4 mm. from the edges of the wound.

On November 26, the wound was very painful and necrosis of the skin had spread to a distance of 6 mm. As this zone became demarcated, part of the necrosis was removed by scissors. The gangrene of the wound continued to spread and on December 7, the wound was treated with 100 Roentgen units. A culture taken from the wound on December 14 showed streptococcus and proteus. Blood examination revealed: Red blood cells 3,900,000 and white cells 11,300, hemoglobin 77 per cent; polys 76 per cent, lymphocytes 17 per cent, eosinophiles 2 per cent, basophiles 1 per cent and mononuclears 4 per cent; 2 plasma cells per 100 white blood cells.

Operation was performed on December 22. The gangrenous zone spreading from the edges was entirely removed by the Paquelin cautery.

Histologic report on the specimen, 12 cm. of necrotic tissue, showed gangrenous fibrous tissues infiltrated with leucocytes, in a state of chronic inflamed infiltration, with purulent ducts and many microbes present.

Aerobic cultures showed proteus; anaerobic cultures showed *Streptococcus putrides*.

The purulent discharge continued and wet zephyrolous dressings were therefore used daily. On December 28, six tablets of sulfanilamide were given. The wound progressed satisfactorily until January 3, when new gangrenous foci at three different points on the medial aspect developed. The electrocautery was used on January 8 and four days later a blood transfusion was given. From January 13 a protracted hypermanganous bath followed by a zephyrolous dressing was given daily. Gangrene, however, spread in all directions. (Fig. 1.)

Fig. 1.

After a consultation by Professors Ádám, Zalka, and Guszman, operation was performed. Far into the intact zone to the muscles, the diathermic knife was carried around the whole necrotic area. Zephyrolous dressings were used daily, with the hypermanganous bath every second day. Twenty units of insulin were given. On February 7 a new focus on the medial aspect was burned out, and five days later a new focus on the lateral side was cauterized into the healthy zone. By February 18 extension had stopped, the wound edges cleared, the bottom of the wound was red, filled with clean granulation, and here and there islands of epidermis appeared. (Fig. 2.)

Thereafter the patient made a speedy recovery and on March 16, apart from a few small areas which had to granulate, the wound was epithelized. The patient was then discharged. The wound was completely healed May 5. (Fig. 3.)

*Pathology.* The necrosis of the wound edges begins slowly, at the beginning involving only small areas—sometimes in areas of deep sutures. An ulcer involves the skin edges which are red, edematous and painful. Soon the necrosis spreads over the connective tissue and deeper layers. The wound edges become elevated, thickened,

and serpiginous in outline, owing to the necrotic process of the subcutaneous tissue, the skin giving a carbuncle-like back from the neck to the buttocks became ulcerated.

Histologically, according to Mayeda, the

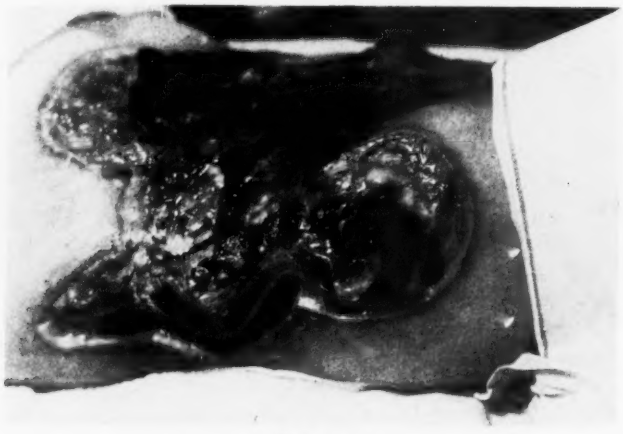

Fig. 2.

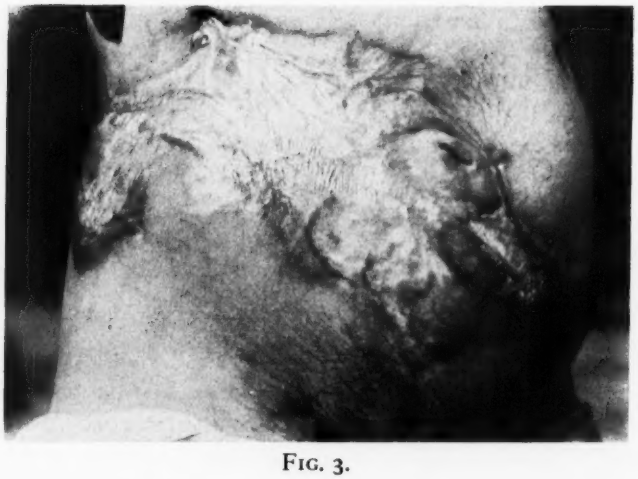

Fig. 3.

picture. The extensive infiltration thereupon changes over to dry type of gangrene. After the removal of the gangrenous zone, a relatively healthy red granulating tissue remains in its place. While the ulcerous process continues, serpiginous healing is to be seen on other areas, sometimes on the isles. The extension occurs slowly, maliciously and usually spreads. Cox distinguishes four zones of extension: (1) outside, painful infiltration; (2) 0.5 cm. sharp demarcated margin of black gangrene toward ulcer; (3) ulcer-ground dirty, grayish-white; (4) well granulating healthy red tissue. The greatest extension observed was in Poate's patient, where the whole necrosis destroys first the border between the papillary and the reticular stratum of the skin. Part of the epidermis and reticular stratum covers the gangrenous mass to a depth of 1.5 cm. and slowly necrotises from the wound edge. A part of the reticular stratum remains intact and in the subcutaneous fat tissue there is some leucocytic infiltration. Where gangrene ends and granulation begins there are a few islands of epidermis arising from remainders of sweat glands which provide a partial basis for later epithelization. According to Wachs' serial histologic examinations, the process is inflammatory and soon leads to necrosis. The infiltration consists mostly of

lymphocytes and greatly surpasses the necrotic border. The skin and papillae quickly fuse, and coagulated fibrin and

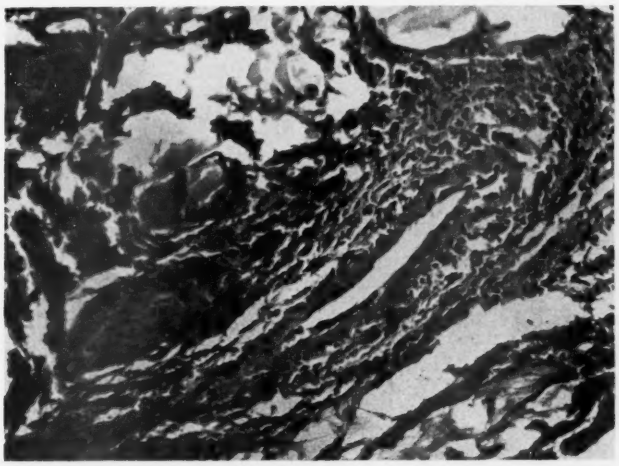

Fig. 4.

lymphocytic infiltration cover the ulcerated area. Nerves and arteries remain intact; the extensive infiltration around the veins is remarkable, even in parts far beyond the inflammatory focus. Many giant cells are present. Thrombophlebitis has been found, not only in the ulcerated zone, but also in the intact area, involving the intima early and later the other coats of the vessel as well. The border is not sharp. Many staphylococci, streptococci and Gram-negative bacilli are to be found in the ulcerated zone.

*Etiology*. In most cases streptococci have been found, and, rarely, staphylococci, diphtheroids or Gram-negative bacilli. Heimburger, Cole and Heideman described cases where the principal role was attributed to the ameba, but this has not been noted by other authors. Most of the patients were debilitated, but no antecedent specific disease, such as lues or diabetes is reported. Lynn attached great importance to the icebag on the abdominal wall as a causative factor, while Borelli attributed the condition to tincture iodine and Tennant to liver insufficiency. In the case of Gatewood and Baldridge, prophylactic scarlet fever serum was given and scarlatina developed. Great doses of serum were then administered and in consequence gangrene of the skin developed on the buttock at the site of injection and spread over the thigh. They believe that the second injection led to gangrene in a hypersensitive organism (Arthus phenomenon). Ballin and Moore supported this view by experiments on animals. However, these various theories have received no confirmation.

The most acceptable etiologic theory is that of Meleney who, after experimentation, explains the development of the gangrene by bacterial synergism, with the streptococcus chiefly involved. Meleney isolated the so-called micro-aerophyl nonhemolytic streptococcus (*Streptococcus evolutus Prévot*), from the extending zone. In the gangrenous zone the streptococcus was associated with *Staphylococcus hemolyticus* and diphtheria bacillus. The diphtheroid bacillus was not pathogenic for animals; nor was the streptococcus or staphylococcus alone, but, when injected together, they caused a process similar to that in humans. Apparently in the extending zone the ground is prepared by the streptococcus, but for the development of the process, symbiosis with other microbes is necessary.

In our case the facultative anaerobic streptococcus with proteus in symbiosis was found. In the cases of Mayeda, Cole and Heideman, Stewart-Wallace and others, necrosis began in deep suture holes where

anaerobic conditions exist. Patterson now omits, for this reason, the use of deep sutures. In our case, however, such sutures were not used.

According to Wachs, thrombophlebitis is the basis of the gangrene and spreads into the intact area. No other author has reported this. In the present case (Fig. 4) the veins showed no change beyond perivascular infiltration. In one of Wachs' cases a varicose ulcer had existed for two years; its sudden spread necessitated amputation of the leg. It is quite possible that thrombophlebitis appeared in the varicose veins and spread, causing a considerable progress of the ulcer. However, it is less probable that this disease had any connection with the skin gangrene. In Wachs' other case, a severe inflammation began in a small infection on the hand. Gangrene spread despite the disarticulation of the fourth and fifth fingers and led to the destruction of the back of the hand as well as a great part of the hypothenar. This case does not appear to have been caused by thrombophlebitis.

It is our belief that the gangrene is due to the direct lytic effect of the microbes.

*Symptoms.* The clinical symptoms correspond to the previously described pathologic-anatomic process. The most characteristic symptom of gangrene of the skin is severe pain, most marked on the edges of the wound. The temperature is usually subnormal, although, with the spread of the lesion higher temperatures may appear. Anemia, exhaustion and mental depression develop with the extension of the gangrene. The process is not contagious.

*Diagnosis.* Diseases which show some similarity to postoperative gangrene, according to Stewart-Wallace, are:

1. Wound infection, where the suppurating infiltration may pass over into necrosis if the cause of infection is especially virulent and the resistance of the patient is low. This might extend to the deeper layer of the abdominal wall, and is the so-called "hospital gangrene" which was not rare before the aseptic period. Skin gangrene differs in that it destroys only the subcutaneous tissue and the skin, not extending to the deeper layer. It is not contagious.

2. Erysipelas in its early stages may be similar, but does not cause a spreading ulcer.

3. Gas gangrene is followed by more serious general symptoms with subcutaneous crepitation and with characteristic bacteriologic findings.

4. Hemolytic streptococcal gangrene of the skin (Meleney) is observed mostly in China, but recently has been seen in New York. This is usually observed on the thigh, sometimes on several places at the same time, with characteristic blood culture and symptoms of exhaustion.

5. Ecthyma gangrenosum is particularly observed in undernourished children and is often multiplex.

6. In specific infarctions, diphtheria, blastomycosis, tuberculosis, etc., the demonstration of the cause of the disease simplifies the differential diagnosis.

7. Amebiasis cutis has been reported only by Heimburger, Cole and Heideman. They believe that a good reaction to an injection of emetin hydrochloride is characteristic, as are the bacteriologic findings.

8. Gangrene may start following human bites, if the deeper tissues are infected by saliva. The presence of fusiform bacillus and spirochetes is characteristic.

*Treatment.* The general opinion is that the only treatment is surgical. Only four cases have been reported as cured without operation. One of these healed in consequence of quartz light (Gordon); one by treatment with 6 per cent sodium chloride (Clinton); one after immunized blood transfusion (Probstein and Selig); and one by the use of maggots (Coakley and Klein). Experiments were made with the local use of various antiseptic and hypertonic solutions, with vitamin diets and creams, with the injection of arsenic, calcium, manganese, antimony, salvarsan, and of specific and nonspecific serums, heteroprotein vac-

cine, with blood transfusions and Roentgen therapy—all without results. The only sure method is the excision of the gangrenous wound edges far into the intact zone, producing healthy wound surfaces. Some authors recommend a knife and some the electrocautery. For styptic action and for cutting off lymph passages, we consider the electrocautery the better.

Little may now be expected from nonsurgical treatment, but we wish to call attention to the use of sulfanilamide which we believe was beneficial in our case. We found also that the use of zephyrolous dressings reduced the quantity of secretion greatly and stimulated the formation of healthier granulating tissue. Blood transfusion may be useful for the recovery of the exhausted patient.

In many cases Thiersch grafts have been used to supply the missing skin. In our patient this was not necessary as the deficiency was soon supplied, partly from the edges and partly from the isles of epidermis in the affected region. It is most interesting that the surface of the wound becomes much smaller and that there is but little contraction of the scar. The cause lies in the sweat glands which remained intact and which supplied some well adhered pinch grafts capable of development.

We consider the 5 per cent mortality of all the collected cases by Lynn to be too low, but the prognosis cannot be called bad if early recognition and suitable treatment are possible. As to prophylaxis, the suggestion of Meleney that deep sutures should not be used on suppurated abdominal and thoracic incisions, we do not deem necessary in view of the rarity of the complication.

## SUMMARY

A case of progressive postoperative gangrene of the abdominal wall has been presented. Hitherto some sixty cases have been reported.

The process is a well outlined entity showing itself in the progressive necrosis of the skin and subcutaneous connective tissues, developing chiefly after operations on suppurating abdominal and thoracic lesions, and produced by symbiosis of specific streptococcus and nonspecific bacteria.

Severe pain is characteristic. The gangrene does not extend to deeper layers and it is not contagious.

Surgery is the only effective treatment. Postoperative treatment consists of sulfanilamide, zephyrolous dressings and blood transfusion. Early diagnosis is important and makes for a better prognosis.

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# ACUTE NECROSIS OF THE KIDNEYS

## REPORT OF A CASE

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BILATERAL cortical necrosis of the kidneys is rare. One of the earliest cases was reported in 1886 by Juhel-Renoy[1] of France. The patient was a girl 16 years old who had scarlet fever and died eight days after the onset of symptoms. Evans and Gilbert[2] added a case to the literature in 1936 and stated in their article that about fifty-three proved cases had been recorded, for the most part by German and English authors. Only fourteen cases have appeared in American medical journals. Evans excluded all cases in which a post-mortem examination was lacking, or when biopsy was done on one kidney only.

In these cases of acute necrosis of the kidneys the etiologic factors were pregnancy (77 per cent), malaria, grippe, scarlet fever, diphtheria, acute perforated appendicitis with peritonitis, acute poisoning, hog cholera, cerebrospinal fever, acute polyarteritis nodosa, pulmonary tuberculosis, pneumonia, carcinoma of the prostate gland, and intravenous camphor.

In most of the cases reviewed there was complete suppression of urine of from two to ten days' duration and death was due to acute renal failure. The uremic phenomena occurred late, usually about the seventh or eighth day of the anuria. Von Zalka,[3] in a review of thirty-one cases in pregnant women, found a uniform clinical picture of eclampsia—convulsions, vomiting, edema, and coma. The urinary output diminished rapidly, with a corresponding rise in the non-protein nitrogen content of the blood. However, in Davidson and Turner's[4] report of four cases in pregnant women there were no convulsions, no suggestion of arteriosclerosis, and no signs of chronic interstitial nephritis. In one case only, post-mortem examination revealed minute areas of degeneration in the liver in addition to cortical necrosis of both kidneys. There were no signs of previous renal damage. In three instances the spleen was congested. Hemolytic streptococci were cultured from the spleen.

In discussing these cases, Morison[5] stated that he had seen this condition only once, when he observed acute necrosis of the cortex of the kidneys in an experimental animal. Saunders and Hartman[6] conducted 100 autopsies on patients who had died of uremia. Several of the kidneys examined showed slight scarring but none presented evidence of acute necrosis.

Because of its rarity and in view of the fact that a search of the literature failed to disclose a case associated with acute pancreatitis, the following case of acute cortical necrosis of the kidneys is reported.

R. W., a white male age 38, was admitted to the hospital October 1, 1937. His illness began five days before entry, on September 26, while he was at the American Legion Convention in New York City. At this time he had what he described as an attack of acute indigestion. He complained of severe upper abdominal pain associated with belching of gas, frequent attacks of vomiting, and diarrhea. On the first day of his illness he noticed blood clots in the urine. The following day he did not void, and no urine was obtained by catheterization. From the onset of the attack he was unable to retain food. Abdominal pain and diarrhea persisted. Therefore he was referred to the hospital.

This patient had had epilepsy since 1918, with some attacks of the grand mal type. Otherwise his past history was non-contributory.

Physical examination revealed a well-developed and well-nourished, but highly nervous man, with marked tremor of the hands. His temperature was 101.8 degrees, his pulse rate 82, respirations 36. His blood pressure was 110/75. There was moderate tenderness in the

epigastrium and over the urinary bladder. The abdomen was distended. There was no edema. Heart and lungs were normal. The fluid was withdrawn. The left ureteral orifice was obscured by blood clot and fibrin.

After this examination the abdominal dis-

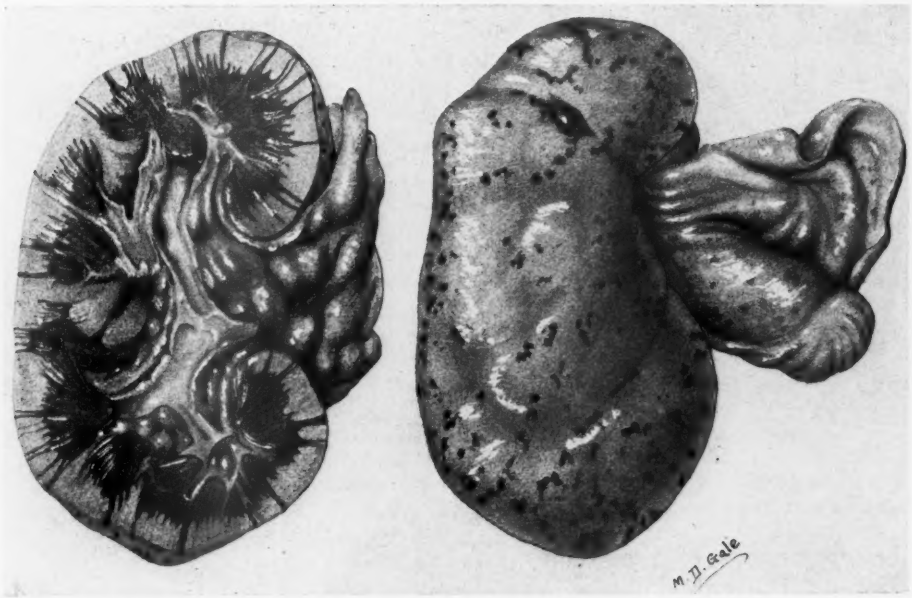

Fig. 1. Drawing of kidney showing gross appearance. Cross-section shows appearance of cortex and medulla. Dark areas indicate the only remaining viable tissue.

white count was 9,900. The red cells showed very slight anisocytosis. The non-protein nitrogen was 171 mg., the creatinine 7.5 mg.

The following day, October 2, a more complete blood examination revealed 3,850,000 erythrocytes, 8,500 leucocytes, and 69 per cent hemoglobin. The differential count showed 80 per cent neutrophiles, 15 per cent lymphocytes, 4 per cent large mononuclears, and 1 per cent eosinophiles. There was very slight anisocytosis.

The blood chemistry was as follows: blood sugar 142 mg., non-protein nitrogen 181 mg., creatinine 8.5 mg.

A flat plate of the renal tract made October 1 showed the kidneys to be normal in size, shape, and position. There was no evidence of renal calculus. A barium enema the following day showed no evidence of large bowel pathology.

On October 2 a cystoscopic examination was performed under spinal anesthesia. A Brown-Berger cystoscope was easily passed into the bladder. The trigone was injected. The trabeculations of the bladder mucosa were prominent. No stones or tumors were seen. A No. 5 catheter was passed up the right ureter for a distance of 25 cm. without meeting any obstruction. A small amount of sanguinous tention decreased to some extent. Two hours later he voided an indeterminable amount of what was considered residual fluid following the irrigation of the bladder. His general condition did not improve. He was treated daily with 3,000 c.c. of 10 per cent glucose administered intravenously and was put in an oxygen tent. His temperature remained between 102.4 and 103 degrees and his pulse rate between 100 and 120.

The blood chemistry determinations were as follows:

| Date | Non-protein Nitrogen, Mg. | Creatinine, Mg. |
|---|---|---|
| October 2 | 181 | 8.6 |
| 3 | 188 | 8.7 |
| 4 | 194.5 | 8.6 |
| 5 | 200 | 8.8 |
| 6 | 207 | 12.0 |
| 7 | 222 | 12.0 |
| 8 | 240 | 12.5 |

The patient gradually became more stuporous and respirations more labored. He finally lapsed into a uremic coma and died on the seventh hospital day, due to complete anuria secondary to an acute nephritis, etiology unknown.

Post-mortem examination revealed an acute pancreatitis with extensive areas of fat necrosis throughout the abdomen and an almost com-

plete necrosis of the cortex of both kidneys. No other etiologic agent was discovered for the anuria except the pancreatitis and its resultant toxemia. The appearance of the kidneys is shown in Figure 1. The right kidney weighed 200 Gm., the left 180 Gm. The cortex measured 5 to 6 mm. The surface of the kidneys showed a number of reddish areas, which represented the only viable tissue in the cortex. The medulla of both kidneys was congested.

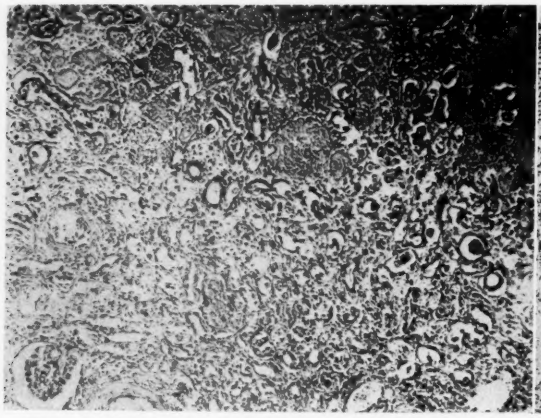

Fig. 2. Section through junction of cortex and medulla showing darker-staining necrotic area and suggestive thrombus in a small vessel adjacent to a glomerulus.

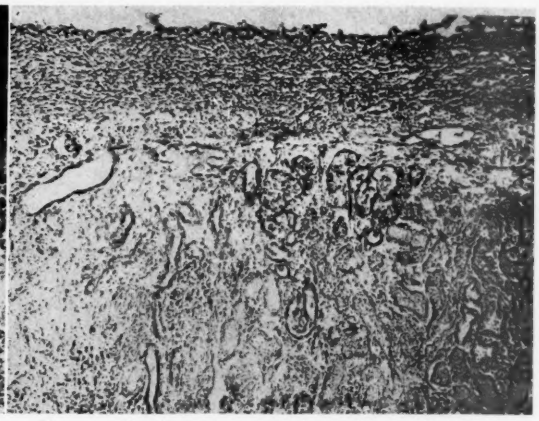

Fig. 3. Section through the subcapsular portion of the cortex showing a small island of tubules which have not been destroyed, probably obtaining their blood supply from the capsule.

Microscopically, the entire cortex showed necrosis of the coagulative or infarct type. At the junction of cortex and medulla there was a polymorphonuclear cell infiltration in the necrotic portion. (Fig. 2.) The involved areas had undergone such a marked change that it was impossible to locate the smaller thrombosed arteries definitely. However, near the junction of the cortex and medulla several larger interlobular arteries were noted. These were filled with necrotic hyaline-like thrombi and the walls of the vessels were also necrotic. Some of the glomeruli at their edges showed only partial or beginning necrosis amid the more normal tubules. Thrombosis of the afferent arterioles was not seen in these early lesions. Beneath the capsule there was a small margin of normal tissue which had probably obtained its blood supply from the capsule. (Fig. 3.)

Thus, the microscopic study revealed acute necrosis of both kidneys and pancreatitis of moderate duration. The fibrous tissue proliferation about the areas of fat necrosis indicated that the first symptoms were due to pancreatitis and that the injury to the kidneys was secondary. The pathology of the kidneys was that of infarction, the injurious agent affecting the arterioles of the cortex. Acute necrosis of the kidneys is secondary to various toxic states, and in this instance the etiologic factor was a pancreatitis of less than usual severity. The blood sugar level was an indication that the pancreas itself suffered only moderate loss of function. No cause of the pancreatitis was elicited. The gallbladder ducts and the pancreatic ducts were patent throughout.

The microscopic descriptions of cortical necrosis which have appeared in the literature correspond with the findings in this case. Many of the glomeruli and afferent and efferent vessels contained fibrinous thrombi. The convoluted tubules contained casts and red blood corpuscles. No endarteritis was seen in the renal arteries and the medulla showed no necrosis.

The question as to where the toxic injury attacks the endothelium is debatable. The glomeruli may be injured first and thrombosis occur in the interlobular arteries; or the arteries may become blocked first with secondary injury to the glomeruli. Jardine and Kennedy[6] in one case found thrombosis only in the capillaries and smaller arteries after two days of anuria. Therefore they concluded that thrombosis began in the

interlobular arteries, not in the glomeruli. Von Zalka, Glynn and Briggs[7] also agreed that thrombosis began in the interlobular arteries, the walls of which became necrotic and showed fibrinous changes. According to their theory, the thrombi moved distally and finally reached the hila of the glomeruli, thus producing necrosis of the cortex. The objection has been raised that if thrombosis began in vessels as large as the interlobular arteries, some changes ought to occur in the spleen. Further study is needed before any conclusions as to the origin of the thrombosis can be reached.

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A PATIENT should be kept under observation for three days after the application of a cast; if swelling occurs underneath the cast, constriction of the circulation resulting in gangrene or contractures may occur within a few hours.

From—"Fractures" by Paul B. Magnuson, 3rd Edition (Lippincott).

# CARCINOMATOUS MASTITIS

## CASE REPORT

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CARCINOMATOUS mastitis is an atypical form of breast cancer which, although very malignant, presents some of the aspects of acute inflammation often so closely as to make differentiation from an inflammatory process quite difficult. It has been alluded to as inflamed cancer, acute brawny cancer, carcinoma mastitoides and mastitis carcinosa. The condition is rapidly progressive and is often improperly treated by fomentations and injudicious incisions on account of the accompanying and confusing inflammatory phenomena. Because of its comparative rarity (1.3 per cent of all breast cancers), this case report is submitted.

Bell, in 1841, first drew attention to this atypical condition. Klotz, Volkmann and Billroth later described it. Schumann, in 1911, reported eleven cases and gave it the name carcinoma mastitoides. He observed that most cases, but not all, arose during late pregnancy or lactation. Lee and Tannenbaum, in 1924, reported twenty-eight cases in detail. They emphasized its distinction from other types of breast cancer and held that irradiation is of value in treatment, being palliative and definitely prolonging life, and is to be preferred to surgery. Fox, in 1930, also stressed the value of irradiation instead of surgery in attacking this condition. Orbach, in 1931, recommended irradiation postoperatively. Chevalier, in 1932, reported a case of acute bilateral carcinomatous mastitis. The extensive involvement precluded any treatment. Hartmann, Bertrand, Fontaine and Guerin (1935) reported ten cases. These were irradiated postoperatively, but the ultimate prognosis was invariably bad. Rubens and Duval in the same year reported six cases. They held that protein therapy combined with irradiation was more efficacious than surgery.

### CASE REPORT

Mrs. G. H., age 33, was delivered of a full term infant on October 21, 1936. About the sixth puerperal day she developed a "lump" in her right breast which was diagnosed "blind boil" by her attending physician who discharged her on the fourteenth day postpartum. The lump subsequently became more painful and was incised under ethyl chloride anesthesia (spray) four weeks later. Some pus was evacuated. Further incisions were made during the following five months but the lesion never healed. The drainage continued.

The patient consulted me on April 7, 1937. The right breast was moderately enlarged, red and hot. The skin was indurated. A large rosette of granulations was present below and to the right of the nipple and a large sinus draining foul-smelling pus penetrated this rosette. Axillary glands were not palpable. A diagnosis of acute suppurative mastitis was made, and under evipal soluble anesthesia intravenously the area was widely incised and drained. Necrotic breast tissue and some foul-smelling pus were evacuated. No histologic study was made at this time.

The patient was discharged in one week, but readmitted on April 24, 1937 for more extensive drainage. Histologic study of the necrotic breast tissue removed was contemplated but the material was inadvertently lost. The patient was again discharged on May 2, 1937. The condition progressed rapidly, the lesion increasing in size and the discharge becoming more profuse. The right axilla presented enlarged, tender, stony hard glands. Mastectomy (Stewart incision) was done on May 12, 1937. The axilla was not explored on account of the extensive infection. May 29, 1937, the incision was practically healed and the patient left the hospital.

Pathologic examination revealed an adenocarcinoma of the breast, grade IV. There was

an open ulcerating area beneath the nipple and several other openings containing a drain. On gross incision there was an open cavity surrounded by firm neoplastic tissue which was granular. Section of the tumor mass showed a thick wall of rapid growing epithelium about the abscess cavity. Many mitotic figures were noted. Acute exudate was found in the stroma and forming the surface of the abscess cavity. Several sections from other parts of the breast showed hyperplasia of the mammary gland with chronic inflammatory infiltration.

Deep *x*-ray treatment with a total of 5600 R was given over the right breast, right supraclavicular and right axillary regions, and over the posterior mediastinum. Generalized metastasis and cachexia supervened gradually. Death occurred in April, 1938.

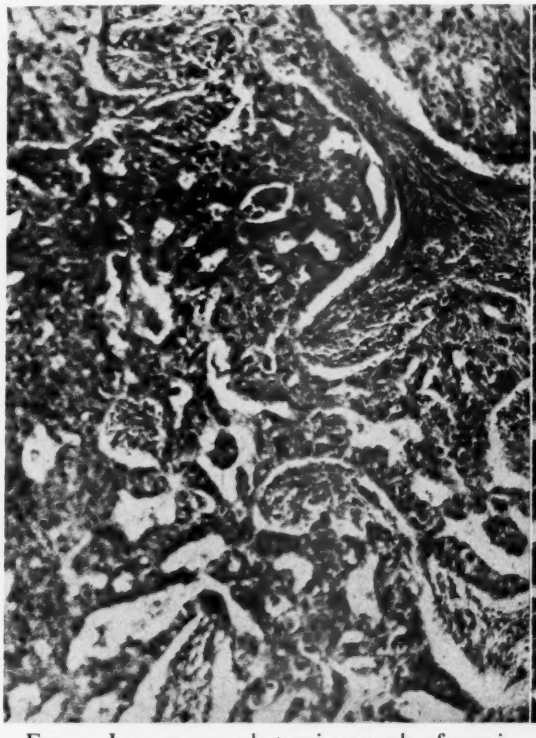

Fig. 1. Low power photomicrograph of carcinomatous mastitis.

Fig. 2. High power photomicrograph.

The marked inflammatory phenomena present in this patient were striking. It was felt at first that the very small incision made under ethyl chloride spray anesthesia was responsible for inadequate drainage and therefore for prolonging the inflammatory process. Only after more extensive drainage was instituted with resultant exaggeration of the lesion were our suspicions of malignancy aroused.

# TRAUMATIC DIABETES

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THE question of the causal relationship of trauma to diabetes mellitus has received considerable attention, and is still an open one. Opinions in the various texts on the subject are often contradictory, and one finds authors denying the possibility on the one hand, yet citing instances in which they are inclined to accept trauma as the cause for the diabetes. It involves the larger problem of neurogenic diabetes, which dates back to Claude Bernard's classic experiment, of producing glycosuria and hyperglycaemia in animals by puncturing the floor of the fourth ventricle. This experiment has never been confirmed in man, but we know that organic disease of the brain such as tumors of the pons, medulla and cerebellum, may produce true diabetes. Dresel and Lewy point to diabetes in cases of organic brain diseases, as tumors or hemorrhages either in the sugar center or near it, or cellular changes in the sugar center.

Joslin dismisses the question briefly, admitting that trauma may aggravate diabetes, but denying a causal relationship. He would discard reports in the older literature because of insufficient proof of previous non-existence of diabetes. He quotes von Norden's very decisive statement based on the experiences of the World War which, he says, once for all bury the question. Yet, in a later discourse, he is not quite so emphatic. Grafe concludes rightly, we feel, that the question must be left to clinical and statistical experiences and that "in a few selected cases the connection between neurogenous and traumatic influences and diabetes cannot be denied."

It is apparent that from a medicolegal standpoint this constitutes an extremely important problem, and it is true that it comes up for debate every so often in litigations concerned with liability and double indemnity insurance. The German courts recognize neurogenous diabetes if the following facts are demonstrable: (1) that there are no facts in the case which would indicate the presence of diabetes before the accident; (2) that the interval between the trauma and the onset be short; (3) that the trauma, especially if mental, be severe.

In a large clinic such as ours one encounters many cases in which the etiology, and particularly the mode of onset, appears obscure, and now and again one obtains a history of trauma, which seems to be definitely responsible for or, at least, a contributing factor in the appearance of the diabetes. Ebstein obtained a history of external injury in six of 116 diabetics, and Williamson in six of 100. In more than half of the cases the head was the site of the injury. There is no proof in any of the cases, however, that diabetes did not exist before the accident. How difficult it is to evaluate such facts is shown by the statements of two authors. Gissel observed hyperglycemia up to 207 mg. for three days following head injuries resulting in coma. In a summary in 1930, R. Stern states that a severe head injury has rarely been followed by a true diabetes.

The pathologic changes described in head trauma are significant, and the onset of diabetes in these cases might be explained by them. Gotten, in a recent review of the subject of head trauma, quotes Winkelman and Eckel, who demonstrated hemorrhages in the brain with subsequent edema, and destruction of brain tissue

around the areas of the petechial hemorrhages. Cassasa described multiple petechial hemorrhages throughout the cortex in patients who had no lacerations of the scalp, nor any fractured skull or contusion of the cortex. Gotten concludes that there is "sufficient evidence that even in cases of mild head trauma hemorrhage occurs, with resultant areas of softening and gliosis of the entire brain." In view of the findings of Dresel and Lewy, mentioned above, a pathologic basis for traumatic diabetes might be established.

We wish to report two cases of diabetes in which trauma played a part.

Case 1. D. M., a white female, age 26, was admitted to the surgical service on March 4, 1936. She had been struck by a street car and thrown to the ground, remaining unconscious for forty-five minutes. She was married, had two children, seven and three years old respectively; there had been no other pregnancies. She had always been obese, and for several months prior to her admission had received injections of antuitrin-s. Her physician informed us that repeated urinalyses proved negative for sugar and that the last analysis was made one week prior to the accident. In November, 1935 (about four months before the accident) she passed a life insurance examination which included urinalysis. Her maternal grandmother had had diabetes.

On admission she was coming out of her stupor, but did not recall the accident. Temperature was 100.6, pulse 110, respiration 26, blood pressure 124/80. There were three extensive lacerations and contusions of the left side of the face. The pupils were equal and reacted normally; no blood or spinal fluid was noted in the aural orifices. Urinalysis was negative. A spinal tap yielded 15 c.c. of frank blood, under normal pressure.

She was given 1,500 units of tetanus antitoxin and 50 c.c. of 50 per cent glucose intravenously. *X*-ray examination of skull and chest proved negative. Neurologic examination showed no extraocular palsies, fields and fundi normal, cranial nerves normal. The diagnosis of cerebral concussion was made.

Two days after admission, seventeen sutures were removed from the scalp, and the wounds were found clean. Urinalysis on March 6 was negative. On March 8, four days after the accident, the patient complained of excessive thirst and frequency of urination. A fasting blood sugar taken that day was 278 mg. This report was overlooked by the surgeons, and the patient was discharged on March 18, with a diagnosis of cerebral concussion and multiple lacerations of the scalp.

After leaving the hospital she noted further polydipsia, polyuria, and a loss in weight of 32 pounds. She felt weak, and consulted her physician, who found glycosuria, and prescribed a diet and insulin. One week prior to her second admission, she became extremely weak, complained of air hunger, and her face took on a reddish-purple color. She was admitted to the medical service on April 16, 1936, twenty-nine days after her discharge from the surgical ward, and forty-three days after the accident.

She was then in deep coma, had Küssmaul breathing and marked acetone odor of the breath. Her temperature was 99, pulse 120, respiration 44, and blood pressure 86/48. A catheterized specimen of urine showed 3 plus albumin, 4 plus sugar, acetone and diacetic acid. The blood sugar was 404 mg. $CO_2$ combining power was not done.

The treatment consisted in gastric lavage, administration of 6,200 c.c. of saline and 5 per cent glucose by hypodermoclysis and venoclysis, and injection of a total of 435 units of insulin during the night. The acetonuria disappeared in five hours, but the glycosuria persisted. The blood sugar reading the next morning was 626 mg. She developed edema, became cyanotic, and died fourteen hours after admission.

An autopsy was performed by the medical examiner who reported the presence of old, infiltrated blood in the deep layers of the galae. The brain was normal on external examination and showed no evidence of surface injuries. The vessels at the base were normal, the ventricles were normal in outline and clear, and there was no evidence of injury or disease in the fourth ventricle. The chest and abdominal cavities were normal. Microscopic examinations of the organs, including the pancreas revealed no abnormalities. No mention was made of microscopic examinations of the brain. The cause of death was given by the medical examiner as "Diabetic coma; diabetes mellitus; laceration of scalp; history of having been struck by street car; accidental."

*Comment.* We believe that the patient, because of her obesity and family history might, in time, have developed diabetes, and that these constituted the predisposing factors, while trauma and shock were the precipitating cause of the disease. She had passed a life insurance examination, and repeated urinalyses made by her physician before the accident were negative for sugar. The actual physical trauma and shock, the cerebral concussion and the hemorrhage, must be accepted as proof of traumatic diabetes in this instance.

Case II. M. T., 14 years old, was admitted to the medical service on January 5, 1938. The family history was negative for diabetes as far back as information was obtainable—the grandparents. At the age of 5, he had sustained a fractured skull and remained in a hospital for four weeks. At the age of 9, he had a second accident with no definite findings, leaving the hospital in good condition after a few weeks. At the age of twelve, he passed a physical examination at school, including urinalysis. Tonsilectomy had been done at 10. There were no other serious illnesses until the summer before admission, when he developed an otitis media which cleared up after two months.

Ten days before admission to the hospital he developed excessive thirst and polyuria and lost 10 pounds in weight. He remained active for about a week, but his mother noted a peculiar flushing of the face. A dose of castor oil given for constipation brought on nausea and vomiting. He became progressively worse and finally stuporous and was brought to the hospital.

On admission he was semicomatose, had acetone odor to the breath, a flushed face, rapid heart, Küssmaul breathing, and a friction rub was heard in the right axilla. (*X*-ray examination of the chest a few days later was negative.) The blood sugar was 390 mg., and the $CO_2$ combining power 21.7 per cent. The urine showed 4 plus sugar, acetone and diacetic acid.

Treatment consisted in administration of a total of 418 units of insulin during the first twenty-four hours, combined with intravenous saline and glucose. The diabetes was well controlled at the end of the second week in the hospital and the boy was discharged on February 1, 1938. He was given a diet of 200 Gm. of carbohydrate, 60 of protein and 75 of fat, with 23 units of protamine insulin daily. A sugar tolerance test was made two days before discharge and the following values were obtained: one-half hour 121 mg.; one hour 358; two hours 348; and three hours 272 mg.

*Comment.* This boy developed diabetes suddenly, after a low-grade infection of two months' duration. He presents a history of trauma on two occasions early in life. There are no predisposing factors, such as obesity or family history. Whether or not trauma played a part in the obscure etiology we cannot say, but it may be assumed that it did. The case is one of many that we encounter, of sudden onset of diabetes with no demonstrable causative factor, and it emphasizes the importance of further study of such cases.

## SUMMARY

Two cases of diabetes mellitus are presented, in one of which trauma appears to have been the precipitating cause, in the other a possible predisposing cause. The relationship of trauma to diabetes is discussed, and a plea made for more careful analysis of all cases of diabetes of obscure etiology, and for reports of all cases of diabetes in which trauma has played a part.

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# INTRALIGAMENTOUS RUPTURE OF A TUBAL PREGNANCY*

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IT is an accepted assumption that 95 per cent of all ectopic pregnancies are caused by low grade infection of the tubes.[1] The other 5 per cent are caused by tumors, agglutination, angulation, blind ostia, etc. When the passage of the fertilized ovum through the tube is interfered with, imbedding will take place in this structure. The stroma in the fallopian tube is scant and does not always undergo decidual changes to resist the digestive action of the trophoblast of the ovum. Consequently, unless the fetus dies, the pregnancy must terminate by rupture of the tube, or by extrusion of the fetus through the fimbriated end into the peritoneal cavity.

After the ovum imbeds itself, the major portion of the growth may be toward the lumen of the tube (luminal imbedding), or toward the wall of the tube (mural imbedding). Some grow equally in both directions (Falk). Near the fimbriated end of the tube, the luminal type of implantation is most frequent. This type is usually operated on as an "unruptured ectopic" or tubal abortion. As one approaches the cornual end of the tube, the mural or combined type is more frequently encountered. It is here that we see the acute catastrophe of a ruptured tubal pregnancy.

When tubal rupture does occur, it may be intra- or extraperitoneal. By far the most common is the intraperitoneal rupture. The incidence of extraperitoneal rupture, or rupture between the layers of the broad ligament is about 1.4 per cent.[2] This is about one extraperitoneal rupture in seventy-five cases of ruptured tubal pregnancy.

Following the rupture of the tube extraperitoneally, the pregnancy may continue unrecognized until term, and a living fetus may be delivered by operation; the fetus may die, with retention of the gestation sac within the layers of the broad ligament for varying lengths of time. Lastly, with rupture of the tube, there may be immediate death of the fetus, with formation of a hematoma in the layers of the broad ligament. This may dissect upward, giving a large retroperitoneal hematoma, or it may rupture secondarily into the peritoneal cavity.[2]

The first intraligamentous rupture of a tubal pregnancy reported by the Harlem Hospital Gynecological Service was seen in April, 1932. The second, which we report now, was seen in March, 1937. During this five year period 149 ectopics were admitted to the service.

J. H., aged 24, was admitted on March 31, 1937, with a complaint of vaginal bleeding of six weeks' duration. Dysuria, frequency, and urgency were noted for four weeks. The patient began to bleed on February 20, which was the expected date for menstruation. The period began normally, stopped after three days, and then started again. Very mild bleeding was noted until the date of admission, with some increase of the amount of flow during the three days in March when the patient expected her period. No cramps, chills, fever, nausea, vomiting or syncope were noted. No previous menorrhagia, metrorrhagia, or dysmenorrhea was reported. The patient had had one child.

General physical examination was essentially negative. Abdominal examination showed slight tenderness in the hypogastrium. Pelvic examination revealed a 2 finger introitus, firm perineum, and a rather roomy vagina. The tip of

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the cervix was softened, the os closed. There was no discoloration. The uterus was anteflexed and slightly enlarged. Hegar's sign was positive. The right fornix was free and non-tender. High up in the left fornix, there was a globular, tender mass, about 4 cm. in diameter. There was slight blood staining of the gloved finger.

Red blood cells numbered 3,900,000, with 70 per cent hemoglobin. Leucocytes were 5,800, with polys 46 per cent, transitionals 6 per cent, lymphocytes 48 per cent. The sedimentation rate was 18 mm. in one hour. A 2 plus Kahn test was reported. An Aschheim-Zondek test was done, but the rabbit died. A second rabbit was not available before operation.

The patient remained on the ward under observation for fourteen days. Bleeding ceased, The abdomen was soft, there was no tenderness, the temperature was normal, and the patient was almost symptom-free. However, she was operated on with the preoperative diagnosis of left ovarian cyst, chronic salpingitis, and left tubal pregnancy to be excluded.

The abdomen was opened through a left paramedian incision. There was no free blood in the peritoneal cavity. The intestines were walled off with laparotomy pads. The right tube and ovary appeared normal, the uterus slightly enlarged. In the region of the left adnexa, there was a large, tortuous, cystic, tense mass, filling the left pelvis. It was deep bluish in color with dilated blood vessels over its surface. The medial end of this cystic blended with the cornual end of the tube. The cystic mass was followed laterally to what appeared to be the fimbriated end of the left tube. It was then noted that the two layers of the broad ligament had been dissected by the dilated left tube. The left ovary was not seen, but it was assumed that it was included in this mass. Adhesions were freed, and during the manipulation the mass was ruptured, spilling forth several blood clots, a live fetus (about three months' size) and a placenta.

The cornual end of the left tube was clamped and cut. The left infundibulopelvic and left broad ligaments were doubly clamped and excised from the uterus by means of a wedged-shaped incision. The wound in the uterus was closed by means of a figure-of-eight suture. All bleeding was controlled. The appendix was not investigated. The peritoneum was closed with a continuous suture, and a one point suspension done at the lower angle of the peritoneal closure. The abdominal wall was closed in layers. No drains were inserted. The patient was returned to the ward in good condition.

The postoperative course was complicated by a mild thrombophlebitis of the left pelvic veins. The patient was discharged on the twenty-fourth postoperative day in good condition. She has since been entirely well.

*Pathologic Examination.* The tube was 6 cm. in length, mildly thickened and tortuous. The ampullary portion measured 1.7 cm. in diameter, and was perforated along its entire length. Spongy friable tissue adhered to the mucosal surface of the tube in the perforated areas. The mesosalpinx and broad ligament showed moderate hemorrhagic discoloration. The fetus was 7.5 cm. long, attached to the placenta by a numbilical cord measuring 10 cm. The placenta was $6\frac{1}{2} \times 4$ cm. and showed small areas of gray and red infarction.

Ruptured ampullar gestation with chronic pseudofollicular salpingitis was the final diagnosis.

## COMMENT

It is of interest to note that both cases of intraligamentous rupture of a tubal pregnancy reported by the Harlem Hospital occurred on the left side. Of interest also is the fact that the patient attended to her usual duties as a domestic up until the time of admission, and the only reason she entered the hospital was because of mild, prolonged, vaginal bleeding.

## CONCLUSIONS

1. The diagnosis of extraperitoneal rupture should be considered when any tentative diagnosis of tubal pregnancy is made, even if the patient presents mild symptomatology.
2. The alteration of the menstrual cycle is a most important symptom in the diagnosis of ectopic gestation.
3. The findings on pelvic examination may be misleading.
4. The Aschheim-Zondek test may or may not aid in the diagnosis.

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# CARBUNCLE OF THE KIDNEY

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THE first description of a carbuncle of the kidney was given by Israel[1] in 1894. The lesion which he noted in the kidney was similar in appearance to a carbuncle on the neck from which his patient had been suffering. Because of the similarity, this observer gave the name, "renal carbuncle," to this pathologic entity.

Moore[2] defines carbuncle of the kidney as a metastatic, circumscribed, conglomerate, suppurative process usually caused by the Staphylococcus aureus, and having its origin in some superficial focus, such as a furuncle, paronychia or carbuncle. The lesion is very frequently limited to the cortex and does not involve the pelvis of the kidney. The typical carbuncle is composed of many small suppurating areas and the entire lesion is separated from the kidney parenchyma by a ring of inflammatory tissue.

It appears that not enough emphasis has been placed upon this condition. There are some who still confuse it with other suppurative conditions of the kidney, or else do not recognize this lesion in the kidney. The acute septic or embolic kidney which shows numerous small abscesses must be separated from the carbuncle, and, in fact, presents a different pathologic picture. There is another condition which must not be confused with carbuncle. This is a metastatic inflammatory lesion found in the cortex of the kidney, caused by the staphylococcus, but which does not show multiple points of suppuration. It may have a necrotic center and an inflammatory tissue reacting wall about it. Many writers have classed these with carbuncles, but some, such as Smirow,[3] call these single, metastatic, staphylococcal abscesses of the kidney cortex, and definitely separate them from carbuncles.

It is generally agreed upon that carbuncle of the kidney is a metastatic lesion from some focus elsewhere in the body. Just how this lesion forms once the organisms arrive in the kidney, is not definitely settled. Some writers believe that a septic embolus is wedged at the point of branching of a small artery, where it is gradually broken down into smaller pieces containing many bacteria. These, then, are carried to different portions of a small area of the kidney with the subsequent formation of many small adjoining abscesses, called together a carbuncle. Barth's[4] conception of the pathogenesis, however, is that a few organisms will settle at one point, multiply

FIG. 1. Carbuncle of the kidney showing numerous suppurative foci with the thickened necrotic capsule visible at the lower pole.

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and by spreading along the lymphatics will produce multiple suppurative areas.

Carbuncles of the kidney are usually unilateral. A few cases of bilateral lesions have been reported. Israel[5] and other investigators explain the unilaterality as due to a lowered resistance of one of the kidneys which has occurred because of injury or previous disease. Schnitzler[6] and Brewer[7] have shown the importance of injury in the production of these lesions. Hunner[8] has demonstrated the rôle which incomplete kidney drainage plays in kidney disease and this may further account for the unilateral occurrence of carbuncle of the kidney.

Renal carbuncles may frequently form perinephritic abscesses. Some investigators believe that all perinephritic abscesses are secondary to this condition. Brady[9] points out that it is characteristic that a carbuncle does not involve the pelvis of the kidney. He states that if the infection has ruptured into the pelvis, the condition is no longer a carbuncle or cortical abscess but a pyelonephritis.

## CASE REPORT

J. M., a white male, aged 18 years, entered the hospital on May 14, 1938 with the history of fever and pain in the left side. The pain, which began one week before admission, was at first diffuse over the entire back and then became localized to the left lumbar region. Three days before admission he developed malaise and a fever of a 102°F. There was no history of chills but the patient did complain of nausea and vomiting. One week before the onset of symptoms, the patient stated that he had had a small abscess over the lateral border of his left wrist which was opened and had healed. The past surgical, medical and venereal history was negative.

Physical examination revealed a well developed and well nourished white male, who appeared acutely ill. The temperature was 101.5°F., the pulse 110 and the respirations 26. The face was flushed, the throat congested. The heart was not enlarged and no murmurs were heard. The abdomen was flat and no masses were palpable. Tenderness and spasm were noted over the left kidney region, but the liver, spleen and kidneys were not palpable. Rectal examination was negative.

*Laboratory Findings.* The urine was cloudy; reaction acid; specific gravity 1.010; albumin, sugar and microscopic examinations were negative. The blood chemistry showed a urea of 16.8 mg. and a glucose of 111 mg. per 100 c.c. of blood. The blood Wassermann was negative. P.S.P. showed an excretion of 85 per cent in two hours. The white blood count was 12,500 with a differential of 82 polymorphonuclear leucocytes and 18 lymphocytes. A pyelogram was done which revealed a definite anterior displacement of the left kidney on lateral view. There was no obliteration of the kidney outline or psoas shadow. Cystoscopic examination revealed a normal urinary bladder mucosa. Excretion from both kidneys was good and approximately equal on both sides. A K.U.B. film showed no abnormalities.

*Clinical Course.* The preoperative diagnosis was abscess of the left kidney with perinephritic extension. A nephrectomy was performed. Following the operation, a wound infection developed and the temperature varied between 99° and 104°F. Transfusions and infusions were given. The patient gradually gained strength, the wound healed slowly and he recovered completely, five weeks after admission.

*Pathologic Report.* The specimen consisted of a kidney (Fig. 1) which measured 11 × 6 × 4 cm. The capsule throughout, especially at the lower pole, was markedly thickened, hemorrhagic and necrotic in areas. The cut surface revealed a mass measuring 4.5 cm. in diameter. It was located in the cortex and the medulla of the kidney. This mass was fairly well circumscribed and yellowish brown in color. A small amount of pus could be expressed from the cut surface. Culture from this pus was positive for Staphylococcus aureus. The surface of the mass had a mottled appearance and numerous small necrotic yellow areas could be seen throughout. The rest of the kidney appeared grossly normal.

Microscopic examination of the mass showed almost complete destruction of the kidney parenchyma by an inflammatory reaction. Here and there the remains of a few glomeruli and tubules were seen. Immediately surrounding the mass the tubules were flattened and many were filled with neutrophiles. The intertubular tissues in this region contained numerous fibroblasts, neutrophiles and many markedly congested blood vessels.

## DISCUSSION

The clinical diagnosis of renal carbuncle presents great difficulties and as a matter

of fact is not often recognized. There is usually a history of some superficial infection with the subsequent development of fever, pain and definite tenderness in one flank. Physical examination may show some muscular rigidity in the upper abdominal and lumbar muscles with moderate but not well localized tenderness. The kidney may or may not be palpable. Leucocytosis is the rule and frequently some abnormality in the urine such as a few red blood cells and a moderate amount of albumin is present. Blood cultures may be positive. Cystoscopic examination may reveal some infection on the affected side with diminution of renal function as compared with the opposite side. The pyelogram may show displacement of the kidney, compression of the pelvis or other filling defects. Intravenous urography may occasionally aid in the diagnosis. Another laboratory test has been found useful. Often, and more so on the left side, due to the extension of the infection to the diaphragm, fixation or limited motion of the diaphragm will occur. *X*-ray or fluoroscopic examinations will allow the examiner to view this phenomenon.

The gross appearance of the lesion is that of a localized necrotic mass within the kidney parenchyma which is of an inflammatory origin. Multiple foci of infection which are demarcated by a fibroblastic wall or which fade gradually into the surrounding tissue without a limiting membrane, may be seen. Abscess formation is late and when it occurs the lesion is preferably referred to as an abscess. The carbuncle is usually located near one of the poles of the kidney and is localized more to the cortex than to the medulla. The process tends to move towards the capsule rather than the pelvis of the kidney.

This is a very serious disease. The sooner the diagnosis is made and treatment instituted, the better the prognosis. The treatment is surgery, which may consist of one of several procedures, such as incision and drainage followed later by nephrectomy, if necessary, primary nephrectomy or excision of the carbuncle. The choice of procedure depends upon the condition of the patient. The lowest mortality appears to be where enucleation is performed but the cases in which this may be done are few. Nephrectomy, in general, appears to be the operation of choice, providing the condition of the patient permits it. When a perinephritic abscess is present, incision and drainage are indicated. Recovery may follow this procedure of drainage without any other treatment being necessary. It is also likely that some carbuncles are never diagnosed and recover spontaneously.

## SUMMARY

A case of renal carbuncle with recovery following nephrectomy is reported.

Carbuncle of the kidney is a definite pathologic entity and should be so recognized.

The gross appearance is similar to that of a carbuncle found on the superficial portions of the body.

It must not be confused with other suppurative conditions of the kidney.

The diagnosis is difficult but if considered, especially when the condition arises following a superficial infection, plus the aid of certain laboratory procedures, it may be more frequently recognized.

The disease is a serious one. The treatment is surgery, the procedure being either incision and drainage, nephrectomy, or excision of the carbuncle. The sooner treatment is instituted, the better the prognosis.

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# MALIGNANT TUMOR OF ADRENAL CORTEX

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KNOWLEDGE concerning adrenal tumors is constantly increasing. In the interest of presenting another example of malignant growth of the adrenal gland this case report is submitted.

## CASE REPORT

Mrs. I. S., age 30, was sent to the Syracuse Memorial Hospital, October 12, 1937. The patient complained of pain in the left upper abdomen, backache and scanty menses. She had been comparatively well until January, 1937, at which time there was a marked lessening of menstruation, a general lack of endurance, increase of weight and an abnormal growth of hair on the face and body.

The patient was married and has one child. Her menses began at the age of 16 and had always been regular, lasting four to five days with a moderate flow. On close questioning the patient stated that the periods may have been shortened earlier. The January period lasted less than two days and no period since had been of greater duration. One menstruation was markedly delayed although no entire month was skipped. Her last period was just before admission to the hospital and lasted only part of one day. The patient has gained between 20 and 25 pounds during one year, and now weighs 193. Her face and body had become covered with hair, enough on her face so that she had been obliged to shave. Her sex desires were normal up to about eight months before, but since January, 1937 there had been an absence of libido. The patient had become nervous and listless.

Examination revealed a markedly overnourished adult female, short and thick set, with coarse facial features. There was an acne-like eruption on the skin of the face, with a rather profuse growth of hair on the upper lip and chin. The voice was coarse and somewhat masculine. The neck was thick and short with no enlargement of the thyroid. The breasts were large and pendulous, the abdomen prominent with a large fat pad overhanging the pubis. The hair on the pubis assumed masculine distribution. A large mass was palpable in the upper left abdominal quadrant, extending over to the midline and down below the crest of the ilium, moving on deep inspiration. There was generalized left-sided tenderness on deep pressure.

The external genitalia were not markedly abnormal, nor was the clitoris hypertrophied. The uterus was normal in size but more firm and fibrous than the uterus of the average woman of 30.

Hypertrichosis of the arms and legs was noted.

The blood pressure was 136/112. The blood count showed 5,000,000 red cells with 80 per cent hemoglobin, leucocytes 8,300, with 83 per cent polys and 17 per cent lymphocytes. The urine on October 12 was negative, except for albumin 1 plus. On the 19th the urine showed 4 plus sugar. A glucose tolerance test was not done. The Wassermann was negative. The N.P.N. was 30 mg. and the blood sugar 93 mg. per cent. Basal metabolism was minus 5 The sedimentation time was 63 minutes.

Cystoscopic examination revealed slight congestion about the left ureteral orifice with a delayed function of the left kidney. A pyelogram revealed the left kidney to be low in position, apparently due to a mass above pressing it downward. *X*-ray examination of the chest was negative. A diagnosis of tumor of the left adrenal was made.

*Operation.* A Cabot incision was made in the left upper quadrant from the xiphoid down the midline nearly to the umbilicus and then laterally to the costal margin. The flap was reflected upward and the abdomen explored. The gall-bladder was soft and the pelvic organs appeared normal. The right kidney was normal and there was no apparent enlargement of the right suprarenal.

The tumor lay in the left upper quadrant with the spleen freely movable above and lateral to it. The left kidney was eventually found below and unattached to it. The tumor itself was soft and about the size of a football.

It had a yellowish-red mottled appearance with many large blood vessels on its surface. With considerable difficulty the tumor was mobilized and the bleeding was controlled. In delivery the growth ruptured, spilling a small mass of friable material into the retroperitoneal space. The tumor was removed and the area drained after closure of the incision in the mesentery of the colon. Throughout the operation the patient received intravenous saline and glucose and at the termination, a transfusion of 500 c.c. of citrated blood was given. The patient returned to her room in good condition.

Twenty-four hours postoperatively, the patient's temperature suddenly rose to 105 with the pulse at 140 and respirations 30. The blood pressure dropped to 95/60, and a few hours later it was 65/40. She had cold, clammy extremities, an ashen appearance and a rapid, thready pulse. Adrenalin was given in ½ c.c. doses every hour for four hours. Caffeine and strychnine were also given as well as fluid by subcutaneous injections. The patient improved under this treatment and was able to take nourishment the following day. Adrenalin was given by hypodermic every four hours for the next forty-eight, at the end of which time she had improved enough to discontinue it. The symptoms of severe shock which she exhibited are very common following the removal of cortical adrenal tumor and many patients succumb at this point. The shock in this case was probably due to postoperative adrenal failure from which the patient recovered. She continued to improve from this time onward and left the hospital on the twenty-seventh day postoperatively, apparently in very satisfactory condition.

*Pathologic Report.* The specimen consisted of a large tumor measuring 19 × 14 × 11 cm. Portions were encapsulated by a thin membrane, which contained a considerable number of blood vessels. The contents extruded from all parts of the specimen but the gross appearance was generally the same throughout. The tumor was soft, friable and broken up into innumerable fine projections. When the mass was bisected there was a definite pattern resembling a cauliflower-like material with a yellow stem. The medullary portion had a distinctly yellow color and was firm in the outer portions, with the center soft, friable, almost necrotic. The outer portion had the appearance of brain tissue.

The tumor cell tended to have rather clear or finely granular cytoplasm, resembling adrenal cortex cells. The arrangement was in rather solid masses with very little evidence of alveolar structure.

Tumor cells were separated from numerous blood spaces by very thin walls or endothelium. Hemorrhage and necrosis were very prominent factors. Mitotic figures were fairly numerous. Microscopically, it was a malignant tumor of the hypernephroma type.

The patient was last seen five months postoperatively. There was no significant weight change. The hair on the face had completely disappeared and that of the abdomen and legs was practically normal in distribution and amount. She had menstruated twice since leaving the hospital and periods were longer but as yet not normal. There was definite pallor, and the red cell determination was 3,600,000, the hemoglobin 60 per cent (Tallquist). There was no evidence at that time of a recurrence of the growth.

In February, 1939 a local recurrence was noted for the first time. This extended rapidly and the patient died six months later.

## COMMENT

The patient who is the subject of this report presented as definitive symptoms of her adrenal pathology the hypertrichosis and masculine escutcheon, coarseness of the skin, increase in weight (20 to 25 pounds in one year) oligomenorrhea, loss of sexual desire and a large abdominal mass. The severe shock which appeared twenty-four hours postoperatively was presumably due to temporary adrenal insufficiency, often observed among postoperative sequelae in this type of case.

# NEW INSTRUMENTS

## A UNIVERSAL ADJUSTABLE STIRRUP FOR IMPROVED LITHOTOMY POSITION*

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OPERATIVE procedures on the perineum require a position which will insure adequate exposure so that

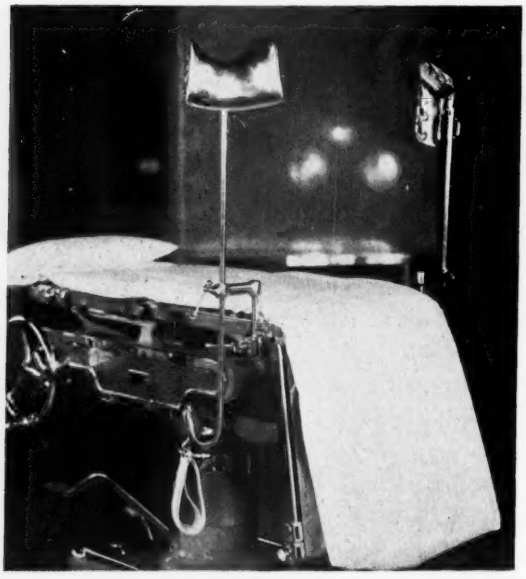

FIG. 1. Stirrup apparatus applied to operating table. Either knee or foot suspension is readily available.

the work can be accomplished with reasonable ease and therefore with a safer and better technique. With the common type of stirrup supplied with most operating tables a sufficient exposure can sometimes be obtained, but not infrequently this proves impossible due to difference between (a) end-width of the operating table and perineal width of the patient, and (b) thigh and leg length measurements in relation to any fixed height of suspension.

In 1907 Young wrote of the importance of a proper position for operative exposure of the perineum for prostatic work, and at that time used the Halsted board with upright stakes. Since then he has devised and described his special table with stirrups for this improved position. Cecil designed stirrups to fit the standard operating table and uses the head of the table to raise the perineum. He discarded the Halsted board in view of the flexibility of the modern operating table equipped with correct stirrups. Because of the difficulty of maintaining a good position for children undergoing cystoscopic examination and eliminating assistants to hold the patient, Campbell fashioned stirrups to fit the standard office examining table for this purpose.

The new type of stirrup here presented may not be superior to the extremity-suspension apparatus described by these authors, for their instruments appear to fulfill the requirements for which they were designed. However, this "universal" stirrup possesses some different features which lend themselves to the various conditions encountered so that the same stirrup may be employed in all instances of lithotomy position.

*Description of Stirrup Instrument.* The instrument is made of a steel alloy which will withstand without bending or turning pressure exerted either by lock devices of

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the table or from muscular efforts of patients during incomplete anesthesia.

The complete stirrup apparatus attached to the operating table is shown in Figure 1, and its component parts of the instrument in diagrammatic fashion in Figure 2. According to the type of suspension desired, whether knee or foot, the straight end of rod B is passed up or down through the slit clamp in the outer end of bar C. Thus rod B may be locked positively at any position with the handle of this clamp and may be adjusted to suit the various heights for different patients, be they short or tall, child or adult. One end of rod D is threaded into the inner end of bar C. The free end of rod D is for connection of the stirrup apparatus to the operating table. It is the only part of the instrument for which specification must be made as to diameter because of the variation in size of slot openings for stirrup attachment in different tables. Since the working distance of bar C is 4 inches on either side, by horizontal adjustment, i.e., locking the stirrup toward or away from the table, the distance between the right and left stirrups may be diminished or increased at any point up to

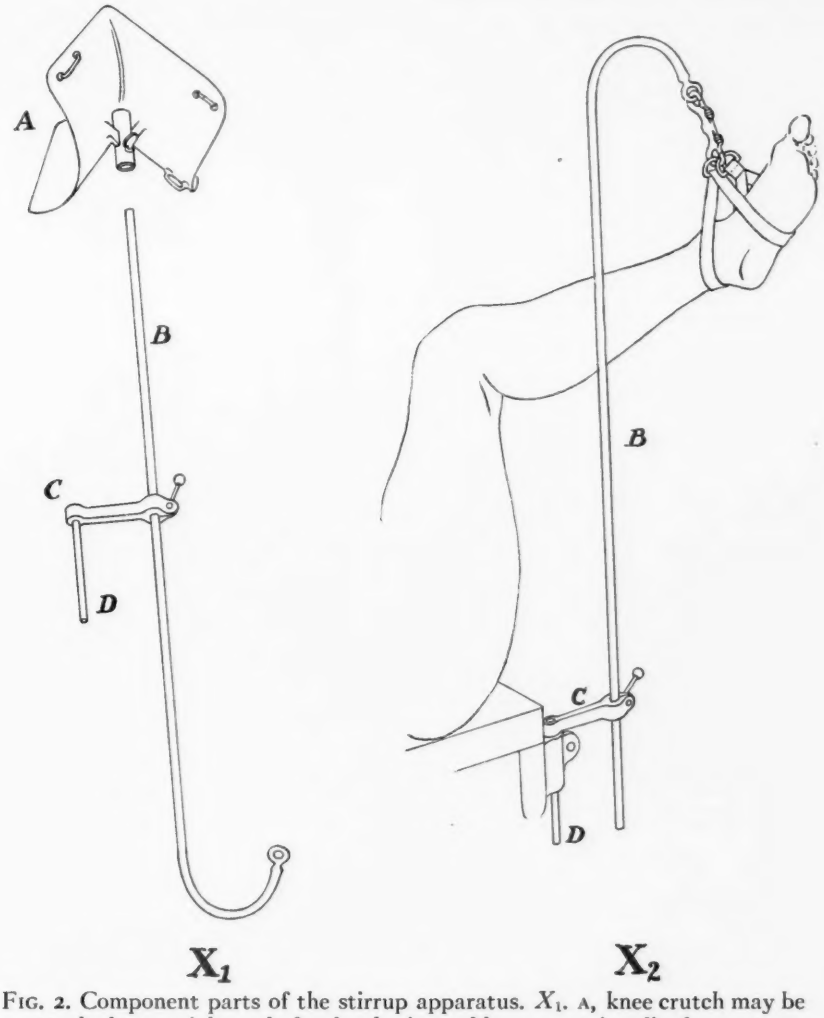


Fig. 2. Component parts of the stirrup apparatus. $X_1$. A, knee crutch may be attached to straight end of rod B; horizontal bar C contains slit clamp at one end through which rod B passes, while into the other end is threaded rod D for attachment to operating table. Rod B is 31 inches long, bar C is 5 inches long and 1 inch thick, rod D is 7 inches long. $X_2$. Stirrup apparatus applied to table for ankle-foot suspension. Buckles and canvas strapping are attached to the cane shaped end of rod B.

8 inches. In this manner a wide or narrow perineum may be brought to or over the end of the operating table without undue

Fig. 3. Application of stirrup apparatus for children. Rod D is rotated inward in front of the table end and locked in position to accommodate perineal width. Rod B is lowered and locked at the proper level to accommodate for thigh and leg length.

stretching of thigh and leg and without impingement of the perineum on the vertical rods of the stirrup. (Figs. 3 and 4.)

When there is insufficient room for first and second assistants so that they must stand behind the patient's thighs, as is the case with many stirrups now in use, their view of the operative field is apt to be obstructed, leading to tiring and inaccurate assistance. In this awkward position, particularly when knee crutches are employed, there is a tendency for the assistants to rest or lean on the patient's legs to reach the operative field, with resultant pressure on the nerves and vessels of the popliteal space. These difficulties are eliminated by providing space for the assistants in front of the patient's thighs. (Fig. 5.)

To be prevented from obtaining a moderate or an exaggerated perineal position because the perineum cannot be

Fig. 4. Application of stirrup apparatus for exaggerated perineal position in adults such as is employed in perineal prostatectomy. Rod D is rotated and locked in extreme outward position, thus increasing the width through which a wide perineum may be brought unhindered over the end of the operating table.

Fig. 5. Showing the added valuable space afforded assistants and operator in front of the operative field.

brought to or over the end of the table; to have the patient's feet swing too closely toward each other or close to the operator's face; to work with insecurely locked stirrups, or those that bend or twist so that the perineum is not maintained in the proper position, definitely increases the difficulty in operating and proves generally annoying.

## CONCLUSIONS

A new and simple type of stirrup is presented which has the following features:

1. Combined foot and knee suspension without changing stirrup apparatus.
2. Made of steel to withstand encountered pressure.
3. Adjustable to correct heights for the various patients so that it can be used on children as well as on any size of adult.
4. Can be used on any operating or examining table having a slot for stirrup attachment.
5. Makes available valuable space for assistants in front of instead of behind the patient's extremities.

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The term "shock" should not be used unqualified. In civilian surgery it should be preceded by the word "surgical" or "traumatic" and in military or air raid surgery the term "wound shock" (1917) is accurate and descriptive. This terminology avoids confusing the condition with "shell-shock" or shock resulting from a transient psychical disturbance.
From—"War Wounds and Air Raid Casualties" (Lewis).

# A NEW INSTRUMENT FOR PASSING PORTIONS OF TENDONS AND FASCIAE LATAE

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THE technical difficulties frequently encountered in the passage of portions of tendons and fasciae latae through bone, stimulated interest in the development of an instrument (Fig. 1) which would facilitate this procedure. The instrument and the technique attendant on its use are particularly applicable to plastic procedures performed in the regions of the shoulder and knee joints, and may be employed further in the transplantation of tendons through soft tissues.

FIG. 1.

FIG. 2.

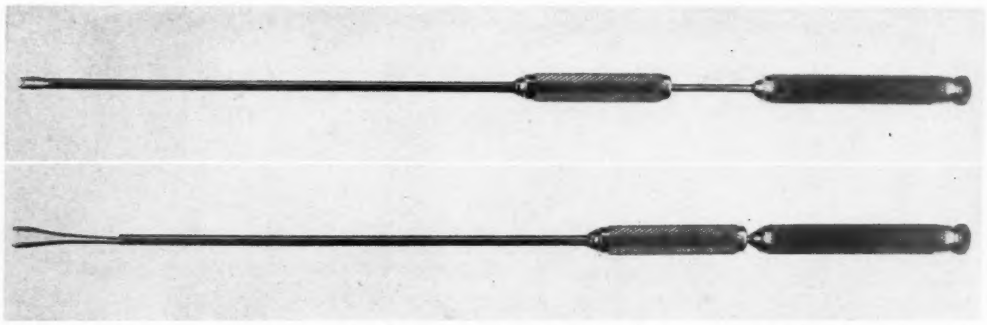

FIG. 1. Tendon and fascia passer in closed position; a forward movement of the closing handle brings the two blades of the forceps together.

FIG. 2. Tendon and fascia passer with blades of the forceps open to receive material to be passed.

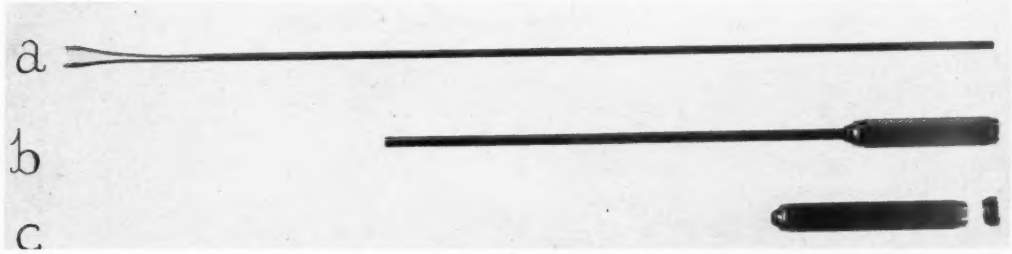


FIG. 3. A, forceps rod; B, tube and handle for closing the blades of the forceps; C, handle with serrated proximal circumference.

The instrument consists of a tissue forceps (Fig. 2) enclosed by a tube; the forceps possesses an unusually long handle having a spike in its serrated mouth. The diameter of the forceps rod (Fig. 3*a*) commonly employed at The Mayo Clinic is 0.125 inches (3.17 mm.), the closing tube (Fig. 3*b*) is 0.135 inches (3.42 mm.) in diameter, and an outside closing tube is 0.191 inches (4.85 mm.) in diameter. There is one handle (Fig. 3*c*) affixed to the forceps rod of the instrument, and another handle, affixed to the tube, for closing or opening the blades of the forceps. Each of these two handles is 0.37 inches (9.40 mm.) in diameter. The over-all length of the instrument is 12.5 inches (31.75 cm.).

[From Fernelius' Universa Medicina, Geneva, 1679.]

# BOOKSHELF BROWSING

## MUSINGS ON THE BILIARY TRACT

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INTEREST in the biliary tract has been manifested since earliest times, beginning apparently in the interpretation of animal sacrifices as a part of divination in which an attempt was made to foretell future events or discover hidden knowledge.

Divination, according to Jastrow,[1] was either voluntary or involuntary. Involuntary divination concerned itself with the understanding of observable facts or events which came to the attention without being deliberately sought. The positions of the stars, the appearances of the clouds and the movements of the planets required some explanation. The birth of a monster and an unusual flight of birds demanded clarification. Much significance was attached to these interpretations.

On the other hand, there was voluntary divination in which one purposely looked for some means by which he might draw aside the curtains and catch a glimpse of an unknown future. In voluntary divination one did not wait to observe in what direction birds were flying or in what formation they were arranged. He did not wait to count their number. Instead, some means at hand was used and an explanation sought. One set loose a flock of birds, observed the direction in which they went, the formation they assumed and the number of birds which followed a definite path.

In the region of the Tigris and the Euphrates several rather detailed systems of divination were produced. After evil had come and the wrath of the gods had descended, people resorted to incantations. Divination had a more purposeful task. This was a method of attempting to change the course of future events or at least preparing oneself for what was to come. Incantations were curative in design; divinations were preventive.

Three systems of divination were perfected in Babylonia and Assyria. One was the inspection of the liver—hepatoscopy. Another was the observation of signs noted in animals at birth. Especially in this second system, the greatest significance was naturally attached to marked abnormalities. The third system was astrology. The movements of the sun, moon, planets and stars—which were all closely identified with the gods—represented the gods actively preparing events shortly to take place on earth.

The inspection of the liver of a sacrificial animal was perhaps the oldest method of

divination. Even before the heart came to be regarded as the center of mental and emotional activity there was, according to Jastrow,[2] "an earlier period in which that distinction was accorded to the liver." Pliny the Elder[3] (23–79 A.D.) mentions the fact that the heart was not used for the purpose of divination until after the 126th Olympiad (274 B.C.). Plato[4] (427–347 B.C.) in the *Timaeus* stated that "the authors of our being, remembering the command of their father when he bade them create the human race as good as they could, that they might correct our inferior parts and make them to attain a measure of truth, placed in the liver the seat of divination."

Why was the liver singled out to be the favorite organ of revelation? Jastrow held that the explanation was a very simple one. Because of the great vascularity of the liver and because blood was associated with life, many primitive peoples considered the liver the center of all mental and emotional activity. It was the seat of life; more than that, however, it was the seat of the soul. Before a journey, a building operation or a military expedition was begun, by the aid of hepatoscopy an attempt was made to gain insight into the future.

Since the gall-bladder was always considered an integral part of the liver because of its very close relationship, it was particularly observed. Peculiarities as to shape, size and position of the gall-bladder were noted in detail. Much attention was directed to the markings on the liver made by the gall-bladder and bile ducts. Observers recalled some past experiences when favorable or unfavorable events followed certain findings. The essential character of the viscus was also considered. Past experience and the association of ideas—these principles determined the interpretations which were made.

What were some of these interpretations? A large, swollen gall-bladder indicated there would be an extension of power; a small and shrunken gall-bladder presaged weakness. In the elaborate system of interpretation which had been developed a sign on the right side always related to the king; a sign on the left side, to the enemy. If the right side of the gall-bladder were tightly embedded in its fossa, surely the king's army would increase in strength. If the gall-bladder were tightly adherent on the right side but loose on the left, it was a favorable sign for the enemy. This observation signified that the king's army would be held in a tight and close grasp. If the left side of the gall-bladder were tightly adherent the indication was that the enemy would be kept as a prisoner and the king's army would be victorious or, at least, the enemy would be kept under control. If the left side showed a tightly embedded viscus, the enemy would be victorious and would hold firmly the king's army in its grasp.

If the common hepatic duct were securely located within the porta hepatis, success would be assured; one not within such protective confines meant an open front to the enemy. A long common bile duct was a most desirable portent because it foretold a long life. The surface markings made by the bile ducts were especially noted by the diviners, and the anomalous conditions which they encountered lent great variety to their interpretations.

Hepatoscopy naturally led to the anatomical study of the liver. Besides the various lobes, the portal vein and porta hepatis, especial interest centered in the gall-bladder and the markings made by the bile ducts. Scribes recorded the interpretations assigned to these variations. Clay models of the sheep's liver were prepared and used for instruction by those who taught in the temples. A picture of such a model now in the British Museum and dating from the time of Hammurabi (2000 B.C.) has been published in "Cuneiform Texts from Babylonian Tablets, etc., in the British Museum." This model is covered with cuneiform writing. It was, in reality, a diagram to explain an omen text. The gall-bladder, portal fissure, cystic duct and part of the hepatic duct are clearly indicated. The gall-bladder, appropriately

enough, bore the name of "the bitter part." The hepatic duct was called "the outlet"; the common bile duct, "the yoke." The porta hepatis was designated as "the crucible."

Almost the same system of hepatoscopy was in practice among the Etruscans. In the Museo Civico at Piacenza is a bronze model of a sheep's liver which dates from the third century B.C. Jastrow was particularly interested in the similarity of this bronze liver of Etruria to the Babylonian counterpart of earlier date which has been described. In the writings of the Greeks and Romans incidental notice is made of Greek and Roman hepatoscopy. There were collections of signs and interpretations of these signs in Roman handbooks but they have not been preserved.

In early zoological literature the gall-bladder in animals was discussed. Aristotle[5] (384–322 B.C.) called attention to the fact that some animals possess a gall-bladder and some do not, and that "among viviparous quadrupeds the stag has none, nor the deer, horse, mule, ass, seal and some swine." Aristotle mentioned the Achainian stag "which appears to have the gall in the tail" and remarked further that "that which they call gall in these animals resembles it in color, but it is not liquid like gall, but more like the spleen in its internal structure."

Pliny[6] was aware of the fact that not every animal had a gall-bladder. He recorded that "at Chalcis, in Euboea, none of the cattle have it, while in the cattle of the Isle of Naxos, it is of extraordinary size, and double, so that to a stranger either of these would appear as good as a prodigy." To Aristotle's list of animals which had no gall-bladder he added the camel and dolphin. He further commented that "some kinds of rats and mice have it. Some few men are without it, and such persons enjoy robust health and a long life." Galen[7] (131–201), the "prince of physicians," had noticed that there were animals in which there was no gall-bladder.

Observations on the gall-bladder have had historical associations as well as those of a religious and scientific nature. Illingworth[8] relates a story which has been told concerning Alexander the Great (336–323 B.C.), the son of Philip of Macedon. When Alexander and his troops returned to Babylon from India, he learned that the sheep which had been sacrificed in honor of his return had no gall-bladder. Alexander considered this a serious omen. In his retreat from India dysentery had taken a heavy toll among his soldiery and he himself was exhausted. Within a few days Alexander also was dead.

Pliny the Elder[9] records an experience of the Emperor Augustus. In the year 32 B.C. the Roman triumvirate came to an end and the Senate declared war on Cleopatra. In 31 the opposing forces met at Actium. Octavian led the land forces; Agrippa commanded the navy. The morning of the battle the Emperor Augustus found a double gall-bladder in the animal he was sacrificing. Great meaning was attached to this discovery, and the overpowering victory of the Roman troops that day made the double gall-bladder seem especially significant to them.

In classical literature one finds references concerning the biliary tract. In the *Electra* of Euripides (480–406 B.C.), Aegisthus, with Pylades for a helper in his task, inspected the viscera of a sacrificed calf. The attention paid to the meaning of the sacrifice is realized as one reads (lines 826–829):

"Aegisthus grasped the inwards
And gazed thereon. No lobe the liver had;
The gate-vein, the gall-bladder nigh thereto,
Portended perilous scathe to him that looked."

It will be recalled that in the *Prometheus Bound* of Aeschylus (389–314 B.C.) the punishment meted to Prometheus was that his liver was to be gnawed upon by a vulture. No more heinous punishment could have been devised in those days. These lines (493–496) are also of interest:

"Smoothness of inwards of beasts sacrificed,
Their hue, which are well-pleasing to the gods,
The gall, the liver's dappled shapeliness—
I set forth the significance of all."

It is not only in the literature of ancient Greece that we find such references. From Chaucer's *Former Age* we recall (line 47) that "Hir hertes were al oon without galles." The absence of the gall-bladder in the pigeon was known to Shakespeare for in *Hamlet* (Act II, scene 2, lines 612–613) the Prince of Denmark himself declares:

"But I am pigeon-liver'd, and lack gall
To make oppression bitter."

The growing realization of the importance of diseases of the biliary tract has been a dominant feature of medical progress in recent years. The methods of study and their far-reaching results are new, but the interest which has developed in this field is only a renewal of one of man's earliest experiences in anatomical observation.

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SPECIAL MONOGRAPH

# THE ESTIMATION OF PELVIC CAPACITY

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# PREFACE

THE present monograph is essentially a collection of ideas concerning the female bony pelvis in its relation to childbearing and, more particularly, of the estimation by pelvimetric methods of its capacity for that function. The application of modern roentgenometric methods to pelvimetry has created a widespread interest in the subject, which has served to emphasize the pioneer work of such men as Michaelis, Litzmann, Breus, Kolisko, and Whitridge Williams of our own time.

The author's interest in this field now extends to a score of years during which time he has written various communications on this subject. A large part of the material here presented has appeared in these writings and credit should, therefore, be given to certain publications in which it has appeared in part. These are *The American Journal of Surgery*, *Journal of the American Medical Association*, *American Journal of Obstetrics and Gynecology*, *Surgery, Gynecology and Obstetrics*, *Yale Journal of Biology and Medicine*, and the author's monograph *The Obstetric Pelvis*. Mention should be made of the courtesy extended by the publishers of the last two publications for permission to reproduce certain illustrations. The author is gratified at this opportunity to record his appreciation of the continued interest and coöperation in his work of his colleagues, Dr. A. H. Morse, Dr. H. M. Wilson, and their associates. Finally, mention should be made of the generous support of the author's work over a period of years by the Research Funds of the School of Medicine, Yale University.

H. T.

# THE ESTIMATION OF PELVIC CAPACITY

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## INTRODUCTION

"Very many be the perils, dangers and throngs which chance to women in their labour." These words, written in 1634 by Thomas Raynalde, the author of the first obstetrical treatise in English, are not out of date, for today, three centuries later, the physician is still seeking for ways to lessen the hazards of childbearing. The dangers to which this early obstetrical author referred, like those of the present day, group themselves into three general categories; namely, those associated with abnormalities of the pregnant state, with puerperal complications, and with mechanical difficulties encountered at the time of parturition. We readily recognize the splendid progress that modern scientific medicine has made toward the solution of these problems, particularly those associated with the first two groups, and during the last decade with the aid of roentgenology new information has given us knowledge which may be applied to the solving of many of the problems associated with the last named group.

Experienced obstetricians will readily agree with Rongy that "Obstetrics is largely a mechanical art. Every case of labor is an engineering problem. The obstetrician, like the engineer, must guide himself wholly in accordance with the principles which make a given mechanical problem safe or unsafe, possible or impossible." Furthermore, it becomes apparent that a knowledge of the contours and dimensions of the bony pelvis is the base upon which the attack on these mechanical problems must be built. We should recall, however, that the successful process of parturition depends not only upon the size and conformation of the bony birth canal, but upon such coördinating factors as the size and position of the fetus, the force and action of the uterine contractions, and the adaptability of the pelvic soft parts.

It is the purpose of this monograph to outline methods which may be employed in ascertaining the contours and dimensions of the bony pelvis in the living subject and to point out the findings that may be expected from such investigation.

"Fortunate it would be indeed for childbearing women" wrote Ramsbotham in 1855, "if they each possessed a pelvis of the figure and dimensions already given as standard. Such, however, is by no means the case; and this organ is subject to great varieties, as well in form as size. It would indeed be difficult to select from all the preserved specimens in existence any two which exactly resemble each other." At a somewhat later date Sir William Turner wrote "with the exception of the skull no portion of the skeleton presents greater individual variations than the pelvis." Roentgenologic studies in our own day have amply proved the soundness of these observations and have further demonstrated that at times apparently minor pelvic variations have a profound effect upon the course and character of labor. In most instances minor pelvic variations do not form insuperable obstacles to labor, but their importance as a factor in labor pathology must not be minimized. There is no question that a large proportion of untimely and ill-advised obstetrical operations are the direct result of a lack of proper understanding of the mechanical aspects of labor in which variations in the contours and the dimensions of the pelvis may play a major part.

Some idea as to the wide range of variability which is found at the pelvic inlet in white women of our population may be gained from the results of a study of 800 adults measured roentgenologically at the Yale Clinic. In 16.1 per cent of this series the transverse diameter was actually shorter than the anteroposterior in this plane. In 45.9 per cent the transverse diameter was no more than 1 cm. longer than the anteroposterior. In 34.0 per cent the transverse diameter was from 1 to 3 cm. longer than the anteroposterior. Finally, in 4.0 per cent this diameter was 3 cm. or more longer than the anteroposterior. As with the pelvic inlet, so also the midpelvis and pelvic outlet show wide ranges in variation. It is obvious, therefore, that if we possess accurate information respecting the size and configuration of these various portions of the pelvis, we shall have an understanding which will be greatly useful in treating many of the problems associated with labor.

It is now nearly a score of years since the author began to study pelves roentgenologically, and so impressive has been the usefulness of the information so obtained that, almost from the beginning, he has recommended the use of Roentgen pelvimetry as a routine procedure in primigravidae. The soundness of this recommendation is being proved with the years of experience, and it is significant that a leader like Munro Kerr within the present year has recorded his

views as follows: "I do not think that, with the knowledge now at hand regarding the influence excited by even minor variations of pelvic formation, we who advocate routine pelvic radiography for all

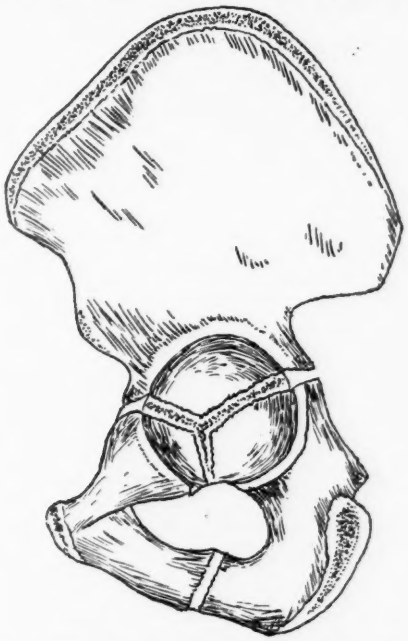


FIG. 1. The three primary centers of ossification unite at the acetabulum about the time of puberty. (From Thoms' "The Obstetric Pelvis," William & Wilkins.)

primigravidae have lost our sense of proportion in this matter, as many suggest today. On the contrary, I make bold to predict that before many years pass it will be accepted as an essential detail of the antenatal examination."

## CONSIDERATIONS OF THE BONY PELVIS

Before mensuration of the pelvis can be discussed, the bony pelvis must be considered in itself. Our knowledge of this structure has been greatly increased during the last decade, but is by no means complete, as is witnessed by the numerous investigations which are now being conducted in this field. In our own clinic male as well as female pelves are being studied in subjects of all ages, ranging from infancy to adulthood, in an effort to learn not only information concerning the incidence of pelvic variations, but something of the factors which combine in their production. The not infrequent occurrence of male pelvic characters in female pelves is a fact which has considerable obstetric significance and which will be discussed later.

It is apparent that there are two sets of factors in operation in the evolution of the pelvis: (1) developmental and hereditary influences; and (2) influences the result of mechanical factors. In the fetal pelvis, long before mechanical factors have been brought into play, sexual differences are readily apparent. At birth the pelvis is both cartilaginous and bony, the innominate bone being in three parts—ilium, ischium and pubis—which are united by cartilage at the acetabulum. The acetabula, iliac crests and ischial rami are almost entirely cartilaginous in their structure. The various parts of the acetabulum are not completely united until after puberty. The pubic and ischial rami unite at about the eighth year. At puberty the secondary centers of the crest of the ilium, the anterior inferior iliac spine (Fig. 1), the symphysis pubis and ischial tuberosity appear, but these structures may not be fused until the twentieth year. Thus it will be seen that during skeletal growth the pelvis is in a relatively plastic state and it is evident that mechanical influences must play an important part in its eventual architecture.

Among these influences may be mentioned the effect of muscular action, the body weight, and the upward and inward force of the heads of the femora. When abnormal plasticity of the bone is present, as in rickets, it is easy to see that marked deformity may thus occur. Schultz, whose studies in primates are fundamentally important, calls attention "to the evident close connection between mechanical factors and evolutionary changes in the primate pelvis, a connection which exists nowhere more pronouncedly than in the pelvis of man."

In considering the capacity of the birth canal it should be remembered that its structure is not only bony but also fibrous and muscular. Furthermore, the bony pelvis is not a definitely fixed structure. Its articulations, which are apparently fixed by strong ligaments, undergo a certain amount of softening under the influence of pregnancy, giving a certain degree of mobility to the pelvic bones. Thus, at the end of pregnancy strong flexion of the thighs on the abdomen, either in the exaggerated lithotomy or squatting positions, definitely, if but slightly, enlarges the pelvic outlet. The well-known Walcher position, where forced extension of the thighs is present, gives a slight enlargement to the pelvic inlet. However, this latter position is so difficult to maintain that its use in dystocia problems is so limited as to be of little practical use. The symphyseal joint separates to a greater or less extent under the influence of pregnancy. At full term this spread averaged 0.58 cm. in thirty-four cases studied by

the author. Rarely the separation may be so marked as to incapacitate the patient, as in the author's reported case. However, the softening of this and other pelvic articulations usually causes but

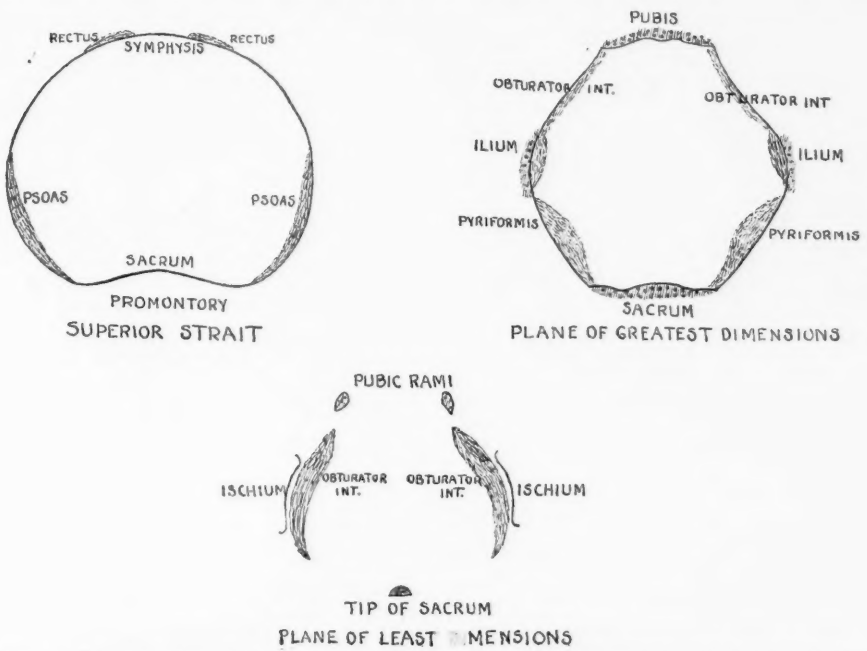



FIG. 2. Diagram showing encroachment of soft parts at different levels of the pelvis. (From Thoms' "The Obstetric Pelvis," Williams & Wilkins.)

little actual enlargement of the pelvis, although in some cases delivery is probably somewhat facilitated.

Certain of the pelvic soft parts may encroach more or less upon the pelvic cavity. In the region of the pelvic inlet the psoas and iliacus muscles protrude slightly as they pass over the pelvic brim (Fig. 2); the rectum if distended may also decrease somewhat pelvic capacity. In the midpelvis the obturator internus and pyriformis project slightly. In the lower pelvis the structures making up the pelvic floor form a barrier at the lower pole of the pelvic canal until this is overcome by the processes of labor. It is, therefore, correct that when we speak of the estimation of pelvic capacity in its true sense we should consider not only bony conformation but these soft structures. However, it is the more or less unyielding bony pelvis which is the chief concern of the obstetrician in estimating pelvic capacity.

The importance of an understanding of the sexual differences in the bony pelvis has been referred to. As early as the third month in fetal life male and female pelves may be differentiated. In the male

fetal pelvis the entire cavity has a more funnel-shaped appearance than in the female, and the sacrosciatic notch in the former is narrower and deeper. According to Thompson "It appears that during

FIG. 3. Front view female and male pelvis showing characteristic differences in pelvic arch.

fetal life the essential sexual characters are as well defined as they are in adult forms, and that any differences that occur during growth between the adult and fetal forms, due it may be to the influence of pressure or muscular traction, affect both sexes alike, and that such influences are in no way accountable, as has been maintained, for the characteristic features of the pelvis as contracted with the male."

In the adult pelvis the sexual differences may be conveniently tabulated as follows:

| Female | Male |
|---|---|
| Greater size, lighter build | Heavier, rougher, more massive bones |
| Greater capacity of pelvic inlet | Lessened capacity of pelvic inlet |
| Greater width of pubic arch | Pubic arch narrower, more angular |
| Larger interischial diameter | Side walls converge, forming a deeper pelvis |
| Lessened convergence of side walls of cavity | |
| Obturator foramen ovoid | Triangular obturator foramen |
| Great sacrosciatic notch, large, almost, rectangular, with longer periphery | Smaller sacrosciatic notch boundaries meet above at acute angle |
| Ilium larger than male | |
| Posterior boundary of sacrosciatic notch chiefly sacral | Posterior boundary of notch sacral only in lower part |

Certain characters of the male pelvis are of definite obstetrical interest because of their occasional occurrence in female pelves. These are: (1) the long narrow sacrosciatic notch and the sacrum in a more forward position which lessens the space between the lower anterior surface of this bone and the ischial spines; (2) the narrow subpubic angle caused by the narrowing course of the pubic rami; and (3) the narrowing of the pelvic side walls from above downward, which restricts pelvic width especially in the mid and lower pelvis. (Figs. 3 and 4.)

The shape of the male pelvic inlet has been described in textbooks almost universally as essentially "heart-shaped" with narrow forepart or retropubic angle and, in comparison with the female, a

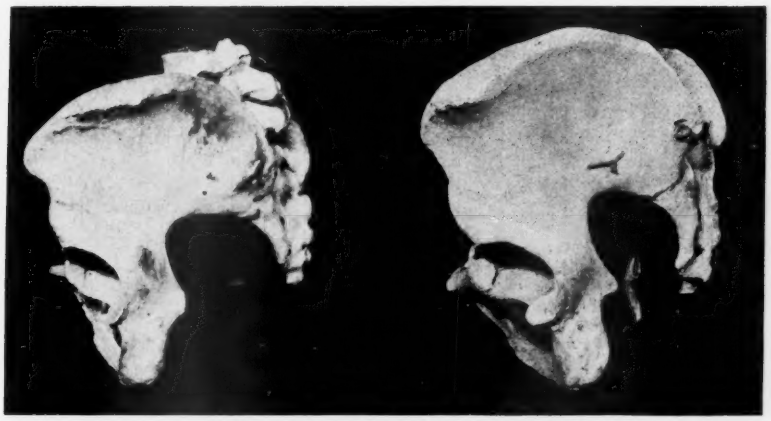


FIG. 4. Lateral view female and male pelvis showing characteristic differences in the sacrosciatic notch and position of sacrum.

posteriorly displaced transverse diameter. These characters have been referred to as typical of the male pelvic inlet and to be quite typical of the "android" pelvis. However, the results of recent investigations by W. W. Greulich and the author do not agree with this concept. (Fig. 5.)

In a Roentgen study of this plane in sixty-nine adult white males of the medical student group, we have found the same essential pelvic variations in pelvic inlet contour which we have found in females, and our conclusions are here repeated.

1. The pelvic bones are heavier than those found in females and, in general, the whole pelvis has a more angular appearance.

2. The pelvic inlet in general appears more circular, and the posterior sagittal diameter in this plane is slightly shorter than that seen in female pelves. This is due to a slight displacement posteriorly of the widest transverse diameter. The forepart of the pelvic inlet differs but slightly from that seen in female pelves and the so-called "heart-shaped" pelvis was not characteristic of the great majority in our series.

3. The constant characters seen were the structural heaviness and prominence of the ischial spines with narrowing of the pelvic side walls from above downward, the angular pelvic arch with relatively narrow subpubic angle and the type of sacrosciatic notch which has been described as characteristically male.

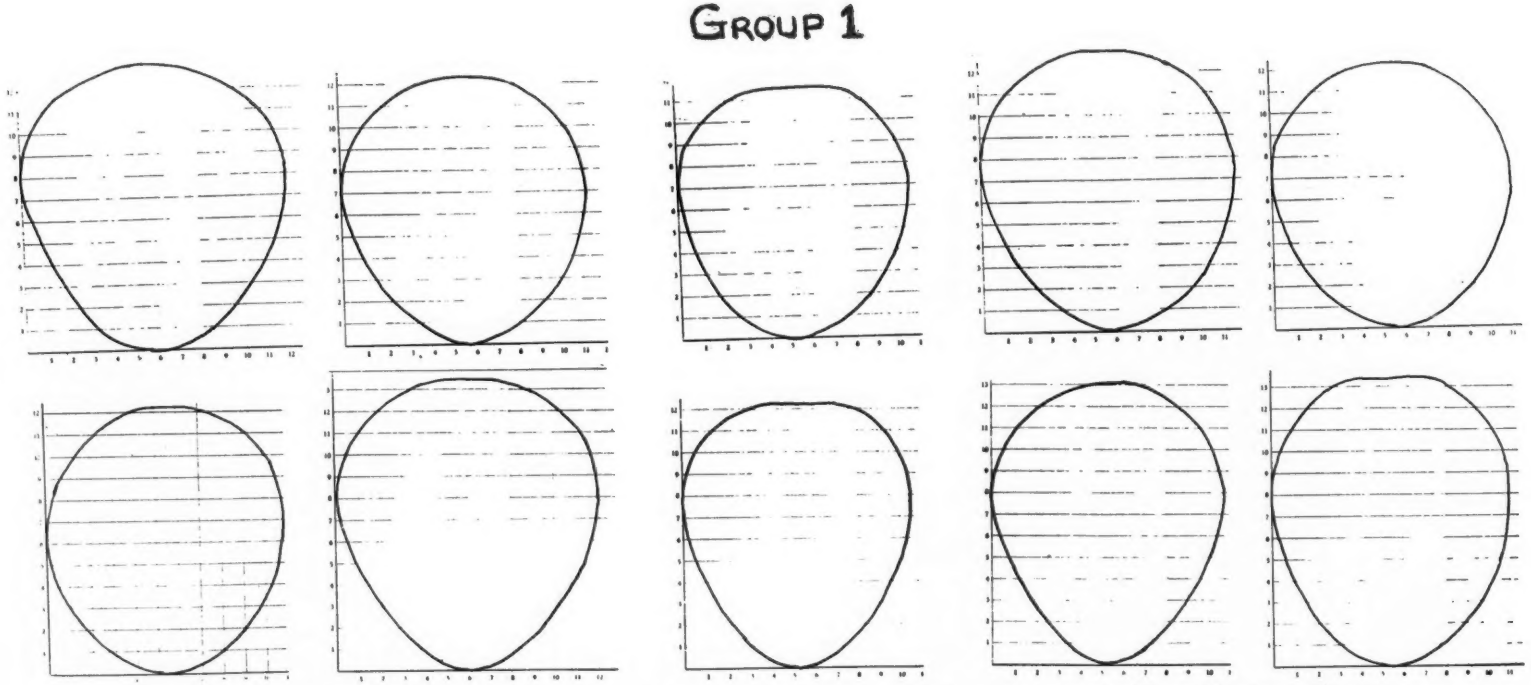



FIG. 5. Specimen tracings of pelvic inlet contours of male pelves showing dolichopellic, mesatipellic and brachypellic types.

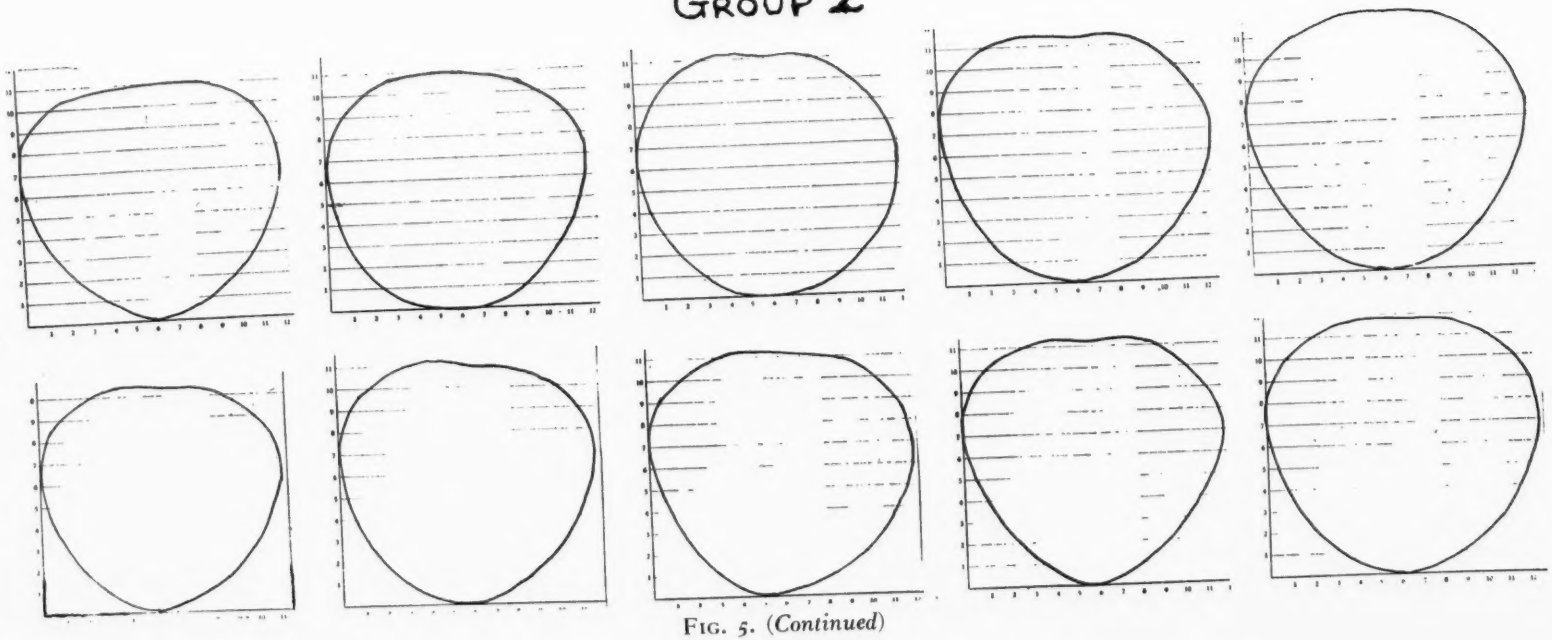



FIG. 5. (*Continued*)

4. The three main pelvic types which we have found in female pelves, i.e., dolichopellic, mesatipellic, and brachypellic were also found to occur in male pelves.

In the foregoing we have discussed the essential differences between male and female pelves, and it is of the highest importance that these facts should be thoroughly understood if we are to intelligently study the obstetrical significance of pelvic capacity.

It is important next to consider the planes and diameters of the bony pelvis which are significant from the point of view of practical obstetrics.

## THE SIGNIFICANT PELVIC DIAMETERS

From the point of view of practical obstetrics there are three portions of the pelvis the mensuration of which will yield useful information. These are: (1) the plane of the pelvic inlet; (2) the midpelvic plane in the lower midpelvis; and (3) the planes of the outlet. A knowledge of the various diameters of these planes will not only furnish a concept of the architecture of the pelvis, but will also give an adequate index of the available space present in the bony birth canal. In addition, this knowledge will enable us to incorporate in the prenatal history a useful and instructive record.

*The Plane of the Pelvic Inlet.* From the obstetrical point of view this plane is bounded anteriorly by the posterior upper surface of the symphysis pubis and the forward portions of the iliopectineal lines, laterally by the iliopectineal lines, and posteriorly by the posterior portions of these lines and the anterior upper surface of the sacrum at the point where the convergence of these lines takes place. It should be noted that this plane is not that of the anatomic superior strait, but rests slightly below this. Its importance in the mechanism of labor has been emphasized, particularly by Caldwell, Moloy, and D'Esopo. In order to simplify the subject, this plane might also be called the obstetrical inlet of the pelvis. The anteroposterior diameter of this plane is referred to by DeLee as the second conjugate. The useful diameters of this plane are (Fig. 6): (1) anteroposterior, (2) transverse and (3) posterior sagittal.

1. The *anteroposterior diameter* of the pelvic inlet extends from a point on the upper posterior surface of the symphysis about 1 cm. from the superior border, posteriorly to the anterior surface of the first sacral vertebra at the point where the iliopectineal lines would

meet if they were to be continued. (This point of convergence may or may not be located at the sacral promontory. It is usually somewhat below this eminence.)

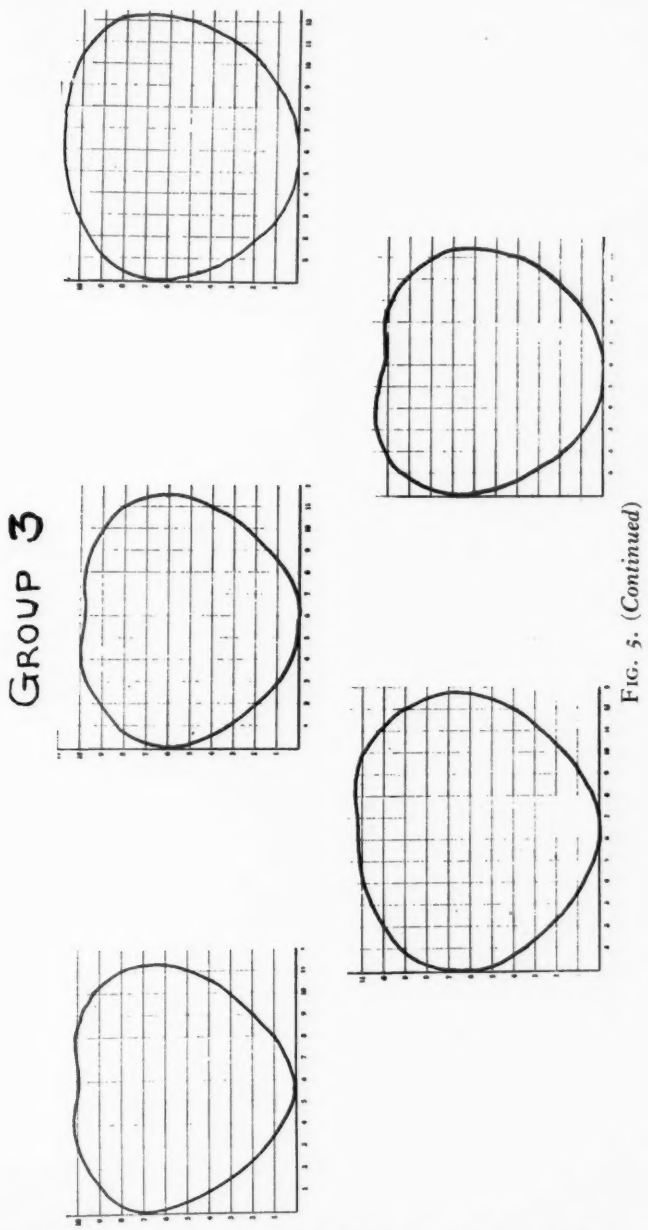



FIG. 5. (*Continued*)

2. The *transverse diameter* of this plane is the widest transverse distance separating the iliopectineal lines. It bisects the anteroposterior somewhat posterior to its midpoint, dependent upon the shape of the plane of the pelvic inlet.

3. The *posterior sagittal diameter* of the inlet is represented by that portion of the anteroposterior diameter which lies posterior to the point of intersection by the transverse diameter. The length of

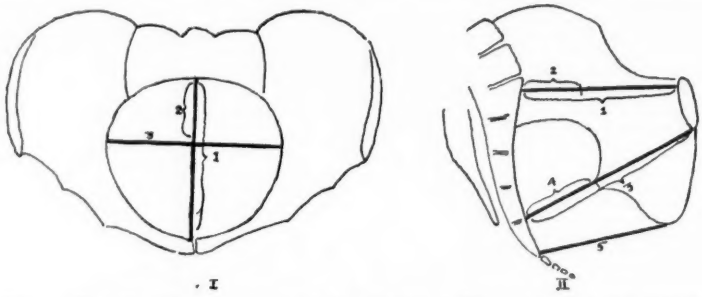


FIG. 6. Significant diameters of the pelvis. *I.* Diameters of the pelvic inlet: 1, anteroposterior diameter. 2, posterior sagittal diameter. 3, transverse diameter.

*II.* Diameters of the lateral pelvis: 1, anteroposterior diameter of inlet. 2, posterior sagittal diameter of inlet. 3, anteroposterior diameter of midplane. 4, posterior sagittal diameter of midplane. 5, posterior sagittal diameter of outlet. (From Thoms and Wilson, in *Yale J. Biol. & Med.*, 11: 179, 1939.)

this diameter is an index of the amount of space in the important upper posterior pelvis and when abnormally shortened usually represents a posterior displacement of the transverse diameter. This may be due to an abnormal convergence of the iliopectineal lines anteriorly, or, in the case of the flat pelvis, to a definite anteroposterior shortening of the inlet of the pelvis. If due to the latter, the discrepancy between the lengths of the anteroposterior and transverse diameters is at once apparent.

*The Midpelvic Plane.* The second obstetrical plane is the midpelvic plane. It is somewhat ovoid in form, large anteriorly and small posteriorly, being narrowed posteriorly by the convergence of the sacrosciatic ligaments. This plane has been defined as being bounded anteriorly by the lower border of the symphysis pubis, laterally by the ischial spines, and posteriorly by the tip of the sacrum. However, as Hanson has emphasized, these points are not truly in the same plane, for the tip of the sacrum is usually as much as 2 cm. below the level of a line joining the lower border of the symphysis with the bispinous diameter. Therefore, the posterior limit should be placed in the lower third of the sacrum and will usually find itself at the junction of the fourth and fifth sacral segments. In our studies of the lateral pelvis aspect, the necessity for this concept of the midpelvic plane has been repeatedly emphasized.

The three diameters which are of obstetrical significance in this plane are the transverse or bispinous, the anteroposterior, and the posterior sagittal.Th is latter diameter, as in the case with the plane

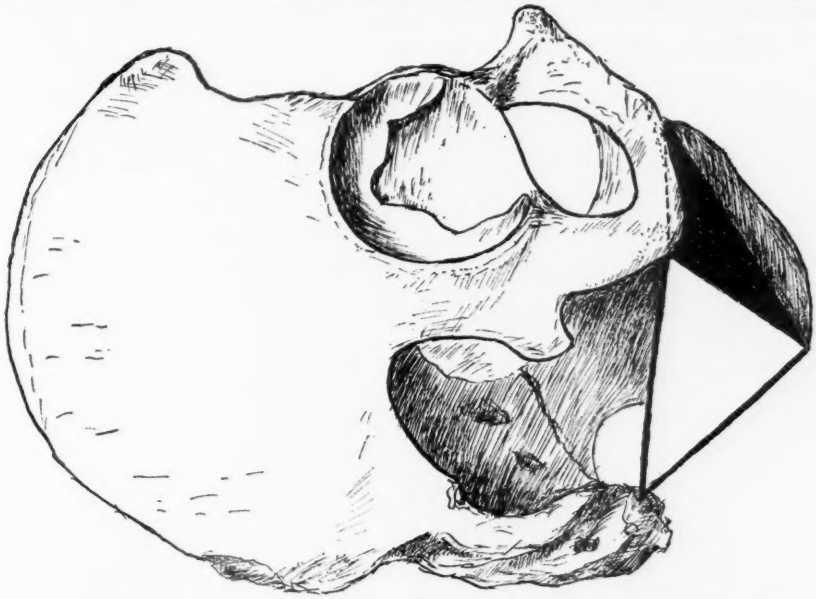


FIG. 7. The planes of the pelvic outlet are essentially two triangles whose bases meet on a line drawn between the tips of the two ischial tuberosities. (From Thoms' "The Obstetric Pelvis," Williams & Wilkins.)

of the pelvic inlet, represents that distance on the anteroposterior diameter which lies posterior to the point of intersection by the transverse or bispinous diameter.

*Planes of the Outlet.* The third portion of the pelvis of definite obstetrical significance is the pelvic outlet. This is in reality two planes—represented by two triangles, the bases of which join each other along the line of the bituberal diameter. (Fig. 7.) The anterior triangle is bounded by the bones forming the pubic arch, the shape of which is of considerable obstetric significance. The two diameters of particular significance are the transverse of the outlet or bituberal diameter, and the posterior sagittal. This latter diameter extends from a midpoint on the bituberal diameter posteriorly to the tip of the sacrum. Other diameters for the pelvic outlet have been described, but do not have essential obstetrical significance and for routine purposes may be omitted.

In addition to the above diameters, all of which may be determined satisfactorily by simple Roentgen methods, there are certain measurements that may be determined by manual methods. These are:

1. The interspinous diameter: The distance between the outer surfaces of the iliac spines.

2. The intercristal diameter: The widest distance between the outer lips of the iliac crests.

3. The external conjugate or Baudelocque's diameter: The distance between the anterior surface of the pubis and the depression under the last lumbar spine.

4. The intertuberal or transverse diameter of the outlet: The distance between the lower innermost surfaces of the ischial tuberosities.

5. The diagonal conjugate diameter: The distance between the sacral promontory and the inferior surface of the symphysis pubis.

The latter two measurements will be considered in more detail later, but the first three have in recent years been shown to be less reliable than was formerly supposed. In 1933, in a paper entitled "The Inadequacy of External Pelvic Measurements," the author compared the results in a Roentgen study of seventy-five pelves. It was pointed out that external measurements must be regarded chiefly as guide posts, for while it was true that small pelves as a rule were accompanied by small external measurements, the reverse was certainly not the case. For instance, in two cases of the series the external conjugate diameter was 20.0 and 16.0 cm. respectively, yet the anteroposterior diameter of the inlet was 10.0 cm. in each. Again in two subjects with identical external conjugate diameters (17.0 cm.) the anteroposterior of the inlet was 7.75 and 11.0 cm. respectively. Students of this subject are therefore in agreement with DeLee in his statement that external measurements are unreliable indices of the size of the pelvic cavity. Of some significance, however, may be the relationship of these three diameters to each other in the case of rachitic pelvis. Possibly for this reason, if for no other, they should be retained.

In addition to the above diameters, which are of great usefulness in determining pelvic capacity, we should mention certain pelvic contours which are also of importance. The shape of the pelvic inlet, the vertical and lateral contours of the anterior sacral surface, the shape of the sacrosciatic notch, the convergence of the pelvic side walls and the prominence of the ischial spines, and the character of the pubic arch are all considerations which should be taken into account. We shall consider these points in greater detail later.

To recapitulate—we can say that the significant diameters for use in the routine survey of the bony pelvis are:

| | |
|---|---|
| Pelvic inlet | Anteroposterior<br>Transverse<br>Posterior sagittal |
| Midpelvic plane | Anteroposterior<br>Transverse or bispinous<br>Posterior sagittal |
| Pelvic outlet | Transverse or bituberal<br>Posterior sagittal |

The above measurements may be considered the cardinal diameters of the pelvis. Of less significance are the diameters known as:

Interspinous
Intercristal
External conjugate or Baudelocque
Diagonal conjugate.

## PELVIC VARIATIONS

From what has been said it is obvious that variations in form of the bony structures of the pelvis show wide distribution. Because of this it has become apparent to all recent students of this subject that the description of the "normal female pelvis" as noted in most modern textbooks requires considerable revision. It is not difficult to understand how an error of this nature has persisted when we consider the relatively limited material which was available to observers in previous times. The use of roentgenologic methods of investigation allows an extensive use of material and it is significant that the occurrence of this textbook type of female pelvis is notable by its relative infrequency. At the present time in white women in this country it is probable that what has previously been described as the normal type of female pelvic inlet occurs in less than one-third of all women.

The intermingling of sex characters and the variability of form of the female pelvis is so great that attempts at classification become most difficult. Some idea of this difficulty may be gained from the statement of Caldwell and Moloy to the effect that "it is difficult to find a pelvis which is typically female in all proportions." The work of these authors in the field is extensive and noteworthy, and the classification which they have suggested, based largely on sex characters, is ingenious and undoubtedly useful for anthropologic study. It is, however, not of easy clinical adaptation because of its complexity. The authors admit that the intermingling of types

renders difficult any attempt to gain simplicity in this method of classification.

It appears to the writer that for the sake of clarity and for direct

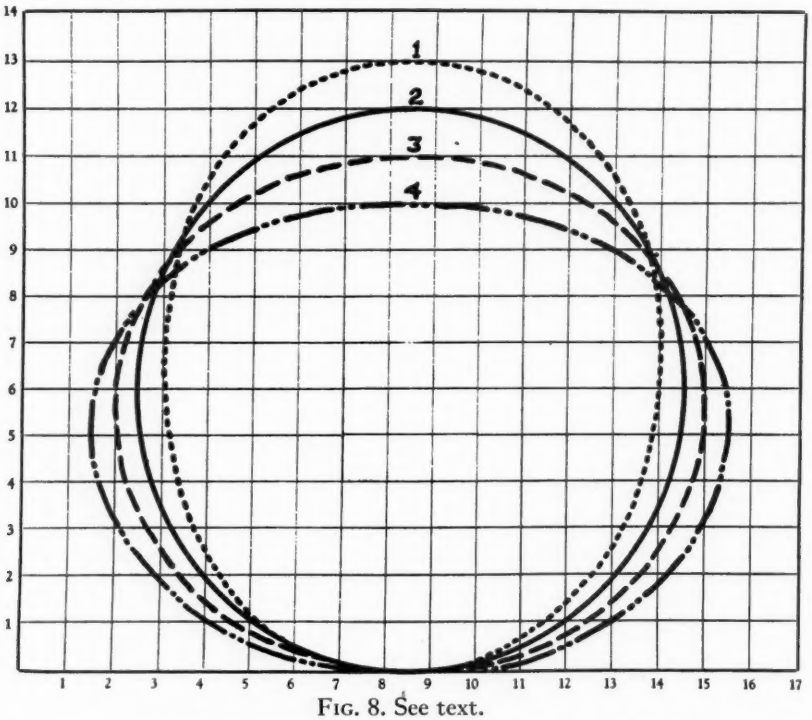



FIG. 8. See text.

clinical application a classification based upon the *general shape* of the pelvic inlet has many advantages. For the past four years we have used such a classification in our clinic and its use by members of our staff over that period has testified to its adaptability to routine work. When it is considered that during this period we have measured the pelvis roentgenologically in every primigravid woman who has been delivered in our clinic, it can be seen that its application has not been inconsiderable.

We consider, therefore, that in general the pelvic inlet may assume one of four shapes (Fig. 8), which may be described as: (1) an anteroposterior oval; (2) a circle; (3) a transverse oval; and (4) an exaggerated transverse oval. We can further describe this morphology by considering the relation of the anteroposterior and transverse diameters of these figures to each other, and on this basis we may adopt terms for pelvic types which will be useful:

1. Dolichopellic or anthropoid type pelvis. The transverse is less than the anteroposterior diameter.

2. Mesatipellic or round type pelvis. The transverse is equal to, or slightly greater (not more than 1 cm.) than, the anteroposterior diameter.

3. Brachypellic or oval type pelvis. The transverse diameter is more than 1 and less than 3 cm. greater than the anteroposterior diameter.

4. Platypellic or flat type pelvis. The transverse diameter is 3 cm. or more greater than the anteroposterior diameter.

The above classification is useful simply as a basis for description of individual pelves; beyond this it does not pretend to venture. After all, in the individual case the obstetrician is not so much concerned with the group in which a pelvis may align itself as he is with the measurements and contours which reveal its useful capacity for childbearing. Given the eight cardinal diameters which have been described, together with a brief consideration of the important contours, such as inlet, notch and pubic arch, one can form a fairly adequate estimation of pelvic capacity for obstetrical purposes.

It might be suggested that we could more correctly use the above terms describing inlet relationships if they were based upon the pelvic index (the ratio of the anteroposterior diameter to the transverse diameter × 100) rather than upon the arbitrary figures chosen. For certain statistical purposes this may be so, but for useful clinical adaptation the use of the index as a basis for classification is cumbersome and does not accomplish any useful purpose. It has also been suggested that the above terms based upon the interrelationships of two diameters may not give a true idea of the shape of the pelvic inlet. This is entirely conceivable but, given the pelvic type plus the knowledge of the three cardinal diameters of this plane, such objection is not well sustained. In any attempt at classification we must bear in mind that if we are to escape being lost among the trees we must keep a perception of the forest ever in mind. With this idea in mind let us recall our original statement that *pelves in general* may be divided into four groups, those whose inlet approximates (1) an anteroposterior oval; (2) a circle; (3) a transverse oval; (4) an exaggerated transverse oval.

It is important to review some of the characteristics of male pelves which may affect female pelves:

1. The male characteristics which may affect the pelvic inlet are: (a) The inlet is in general more circular. (b) The widest transverse

diameter is, therefore, slightly displaced posteriorly. (c) The posterior sagittal diameter is relatively shortened.

2. The male characteristics which may affect the midplane are: (a) The narrowing of the pelvic side walls with increase in size and prominence of ischial spines results in a relatively decreased bispinous diameter. (b) Laterally the male type or narrow sacrosciatic notch is present which decreases posterior pelvic capacity in this plane and manifests itself in a relatively shortened posterior sagittal diameter.

3. The male characteristics which may affect the pelvic outlet are: (a) Narrowing of the pubic arch is present, with less arcuate formation of its sides and a relative shortening of the bituberal diameter. (b) Because of the forward displacement of the sacrum forming the posterior wall of the male notch, the posterior pelvic capacity in this plane is also decreased, which manifests itself in a relatively shortened posterior sagittal diameter of the outlet.

These male characters may appear in female pelves at the inlet, the midpelvis, or the outlet, and in a certain small group of pelves they may occur in all three portions, the complete male pelvic type. It is obvious that they may have a considerable obstetrical significance, particularly in the latter instance.

Some idea of the distribution of these basic types in white women of the clinic group may be gained from their incidence in 800 primigravidae who were delivered at term (child 2500 Gm. or over) in the New Haven Hospital. (Table 1.) Again the fact impresses

TABLE 1

| Type | Number | Per Cent |
|---|---|---|
| Dolichopellic | 129 | 16.12 |
| Mesatipellic | 367 | 45.88 |
| Brachypellic | 272 | 34.00 |
| Platypellic | 32 | 4.00 |

us that 62 per cent of this series present pelves whose inlet is not that described as normal in textbooks of anatomy. In a recent study by Greulich and Thoms of the pelvic inlet in 789 white females, which included 582 clinic women, 100 nurses, and 107 young females from 5 to 15 years, the incidence of pelvic types was as indicated in Table 11. The incidence of the various types in the student nurses

seems especially significant in view of the superior physical status and economic level of the women comprising that group. The data from the younger girls is also of significant interest, showing the high incidence of the dolichopellic type in this group.

TABLE II

| | 100 Nurses | 582 Clinic women | 107 Children |
|---|---|---|---|
| Dolichopellic | 37.0 | 14.5 | 57.9 |
| Mesatipellic | 46.0 | 44.5 | 33.6 |
| Brachypellic | 17.0 | 34.3 | 8.3 |
| Platypellic | .... | 6.7 | |

Our conclusions follow:

1. The type of pelvis which for the past two centuries has been considered normal for white women was found in less than 15 per cent of 582 primiparous clinic patients, and in only 6 per cent of 100 young women from a much more privileged economic group. It was, therefore, neither the normal pelvis—in the sense of being the most frequently occurring type—nor was it the most adequate type, as gauged by the relative frequency of operative intervention required during labor by the women possessing it.

2. It has long been known that marked anteroposterior flattening of the adult pelvis may result from severe rickets during early life. The high incidence of round and of anteroposteriorly elongated pelves among the student nurses of our series suggest the possibility that adequate nutrition during early life and other factors which make for the attainment of maximum normal body size prevent that degree of anteroposterior flattening of the pelvis, which has come to be considered as characteristically feminine.

In the series of 800 white clinic women from whom the statistics quoted above were taken, the entire pelvis was not surveyed roentgenologically because the first part of the group antedated our practice of taking routine lateral views. However, in the latter 200 of this series this was done and in this group the incidence of pelvic type followed the same general trend as in the larger group. (Table III.)

In order to gain some idea of the normal range of measurements, the mean values of the various diameters are given. (Table IV.) From a survey of this table certain conclusions may be drawn. In the dolichopellic type there is a tendency for the anteroposterior-

transverse relationship to be maintained throughout the pelvis. In the brachypellic type a similar tendency is maintained, although in a less striking manner. In the platypellic group the shortened posterior sagittal diameter of the inlet and wide bispinous diameter suggest that rickets plays a rôle in the etiology of this group and evidences of the disease in a distortion of the contours and position of the sacrum will be found in many instances.

TABLE III

| Type | Number | Per Cent |
|---|---|---|
| Dolichopellic | 37 | 18.5 |
| Mesatipellic | 95 | 47.5 |
| Brachypellic | 62 | 31.0 |
| Platypellic | 6 | 3.0 |

TABLE IV

| | Inlet | | Midplane | | | | Outlet | |
|---|---|---|---|---|---|---|---|---|
| | A.P. | Trans. | P.S. | A.P. | Trans. | P.S. | Trans. | P.S. |
| Dolichopellic | 12.53 | 11.72 | 5.07 | 12.55 | 9.45 | 5.22 | 8.95 | 7.84 |
| Mesatipellic | 11.75 | 12.32 | 4.48 | 12.34 | 10.34 | 5.23 | 9.16 | 7.71 |
| Brachypellic | 11.06 | 12.67 | 4.15 | 12.01 | 10.32 | 5.23 | 8.92 | 8.05 |
| Platypellic | 9.00 | 12.67 | 2.75 | 11.67 | 10.45 | 4.71 | 9.12 | 7.58 |

For purposes of clinical comparison it may be useful to establish criteria for the designation of *small pelves* in each group. Such a subdivision is necessarily arbitrary. However, if "average" pelves may be defined as those lying within the interquartile range, that is in the middle half of all, then small pelves may be defined as those which are measurably smaller. If we use the anteroposterior diameter of the inlet as the yardstick, we find that for the first three pelvic types the lower limit of the average group is 0.5 cm. less than the mean for the type. About one-fifth of all pelves are measurably smaller and may, therefore, be designated as small. This being so, small pelves occur with approximately equal frequency in the three pelvic types, as indicated in Table v.

It may be clinically useful to study the mean measurements of this group of 200 pelves in white women and discuss the lower limits of normal for each measurement. By this means a table may be made giving us these limits of which we can say that dimensions

smaller than those given for each diameter should be viewed circumspectly. (Table VI.)

TABLE V

| Type | Anteroposterior Diameter | Number | Per Cent |
|---|---|---|---|
| Dolichopellic | Less than 12 cm. | 7 | 18.9 |
| Mesatipellic | Less than 11.25 cm. | 17 | 17.9 |
| Brachypellic | Less than 10.5 cm. | 11 | 17.7 |

TABLE VI

| | Inlet | | | Midpelvis | | | Outlet | |
|---|---|---|---|---|---|---|---|---|
| | A.P. | Trans. | P.S. | A.P. | Trans. | P.S. | Trans. | P.S. |
| Dolichopellic | 12.0 | 11.25 | 4.5 | 12.0 | 9.25 | 5.0 | 8.5 | 7.5 |
| Mesatipellic | 11.25 | 11.75 | 4.0 | 11.75 | 9.75 | 5.0 | 8.5 | 7.5 |
| Brachypellic | 10.75 | 12.25 | 3.75 | 11.5 | 10.0 | 5.0 | 8.5 | 7.5 |

*Clinical Relationships.* In a recent paper the author has analyzed certain clinical relationships of the pelvic variations of the above group of 200 pelves and some of these statistics are of considerable interest. Thus in this group of primigravid women consecutively delivered at term (child 2500 Gm. or over) the labor was terminated by operative intervention thirty-six times, or 18.0 per cent. An analysis of this intervention is:

Dolichopellic Type

1. Small pelvis—contracted midplane and outlet—mid forceps.
2. Small pelvis—contracted midplane—low forceps.
3. Child 3920 Gm. arrest at outlet—low forceps.
4. Child 4175 Gm. arrest at outlet—low forceps.
5. Prolonged labor—contracted outlet—low forceps.
6. Persistent posterior position—low forceps.
7. Prolonged labor—low forceps.
8. Small pelvis—contracted midplane and outlet—low forceps.

Mesatipellic Type

1. Persistent face presentation—cesarean section.
2. Premature separation of placenta—cesarean section.
3. Posterior arrest in midpelvis—mid forceps.
4. Contracted midplane—mid forceps.
5. Child 4180 Gm.—low forceps.
6. Arrest at outlet—low forceps.
7. Small pelvis—prolonged labor—low forceps.

8. Arrest at outlet—low forceps.
9. Small pelvis—low forceps.
10. Prolonged labor—low forceps.
11. Contracted midplane—low forceps.
12. Persistent posterior—low forceps.
13. Small pelvis—low forceps.
14. Outlet contraction—low forceps.

Brachypellic Type

1. Hypertension—dystocia syndrome—cesarean section.
2. Premature separation of placenta—cesarean section.
3. Contracted midplane—child 3935 Gm.—mid forceps.
4. Transverse arrest—contracted outlet—mid forceps.
5. Small pelvis—contracted midplane—mid forceps.
6. Prolonged second stage—child 3980 Gm.—mid forceps.
7. Contracted midpelvis and outlet—child 4135 Gm.—mid forceps.
8. Rheumatic heart disease—mid forceps.
9. Arrest in midpelvis—inertia—mid forceps.
10. Inertia—low forceps.
11. Contracted midpelvis—low forceps.
12. Contracted outlet—low forceps.
13. Small pelvis—contracted outlet—low forceps.
14. Contracted midplane—low forceps.
15. Pulmonary tuberculosis—low forceps.

Platypellic Type

1. Rachitic pelvis—cesarean section.

Omitting outlet or low forceps in this series of 200 deliveries, we find that operative intervention was done in fifteen instances, or 7.5 per cent of cases. These were distributed as follows:

Dolichopellic type—1 operation—2.5 per cent.
Mesatipellic type—4 operations—4.2 per cent.
Brachypellic type—9 operations—14.5 per cent.
Platypellic type—1 operation—16.6 per cent.

These latter figures correspond very well with the operative intervention in a series of 600 cases of delivery in white women at term which were previously reported, and serve to emphasize further our conviction that the most favorable type of pelvic inlet is that which is round or is elongated anteroposteriorly (mesatipellic or dolichopellic) and not the oval (brachypellic) or "textbook" type of pelvis.

In this series of 200 deliveries labor was prolonged (24 hours or more) in thirty-eight instances (19.0 per cent). In eleven of these cases the outlet was contracted, in fourteen the inlet showed dimensions less than normal for the type, and in five the midplane showed contraction, either alone or in combination with some other portion of the pelvis. All of these findings show the importance of an accurate

pelvimetric survey during the antenatal period, and make it apparent that unless primigravid patients are routinely thus examined, many significant changes in the bony pelvis will remain undiscovered.

The subject of pelvic variations is a particularly fascinating subject, not only because this newer knowledge has opened up a wide field for investigation, but also because of the importance of this knowledge to the function of childbearing. It is obvious that if a mammalian species is to survive, the pelves of the females of that species must be large enough to permit the birth of their young. Because of such relationship, anthropologists of the nineteenth century were led to expect a rather close correspondence between the shape of the head and the shape of the pelvic inlet in various races. And today among certain obstetricians there is an opinion that rather close relationships exist between pelvic type and body build. In a recent paper entitled "A Study of Pelvic Type," Greulich and Thoms have reported the results of a study which attempted to investigate such relationships. We measured and photographed 132 of the clinic women and 104 student nurses, all of the latter being college graduates. Some of the relationships between pelvic type and body build in the series are here summarized.

1. The clinic women and student nurses who had dolichopellic and mesatipellic pelves were on the average the tallest women of our series, those with a relatively wider pelvic inlet, the brachypellic type, were the shortest.

2. The clinic women had relatively longer trunks and shorter legs than the student nurses of corresponding pelvic type.

3. The women with long oval pelves (dolichopellic) were predominantly tall, long-headed and broad-shouldered. The width of their pelves between the iliac crests and of their hips between the trochanters was smallest in proportion to the width of their shoulders in comparison with women of the other pelvic types, and they had the largest external conjugate diameter.

4. The women with the transversely elongated pelves (brachypellic) were, on the average, the shortest of the series, and they had the broadest heads, the narrowest shoulders, the widest pelves and hips in proportion to the width of their shoulders and the smallest average external conjugate diameter.

5. The women with round pelves (mesatipellic) were approximately intermediate between the other two groups in all these dimensions.

6. There was much variation in these external dimensions between student nurses and clinic women of the same pelvic type and it is our opinion that to attempt to predict pelvic type on the basis of external dimensions in individual cases is quite hazardous.

7. The high incidence of dolichopellic and mesatipellic pelves among the largest women of both groups suggests the possibility that nutritive and other factors which make for the attainment of maximum normal growth tend to prevent anteroposterior flattening of the pelvis, such as is noted in the brachypellic and platypellic types.

8. Finally, our observations indicate that the type of pelvic inlet can be determined in the intact living women only by roentgenographic means.

## THE ESTIMATION OF PELVIC CAPACITY

Modern methods for measuring and estimating pelvic capacity may be divided into two groups: (1) external and internal palpatory methods; (2) roentgenologic methods. An outline-chart of the procedures used in our clinic is shown in Figure 9. This is known as the Pelvic Estimation Chart, and it is filed with each patient's prenatal record in order to be available as soon as she enters the hospital for delivery. It is my conviction that such data, together with a brief comment, will furnish information concerning pelvic capacity which is adequate for all ordinary obstetric purposes. It might be useful to consider in order some of the details of examination that are noted in this outline.

*External Measurements.* As stated before, the information gained from these external measurements should be considered solely from the standpoint of the guide post. Small external measurements are usually found in patients with pelves of small capacity, but the reverse is by no means true. When the external conjugate diameter is relatively shortened, the probability of anteroposterior shortening of the pelvic inlet should be considered. While the interspinous and intercristal diameters give but little information concerning pelvic capacity, nevertheless their relation to each other may be of significance. In certain cases of rachitic pelvis, owing to the outward flare of the iliac bones, the normal relationship of these two measurements may be reversed, the intercristal diameter being shorter than the interspinous.

NEW HAVEN HOSPITAL AND DISPENSARY—PELVIC ESTIMATION CHART

Name_____ Date_____ No._____

**External Measurements**

Spines .....cm. Crests.....cm. Ext. Conj.....cm.

Pubic Arch Wide Average Narrow

Rami Arcuate Straight

Bituberal..... cm. Post Sag.....cm.

**Rectal and Vaginal Examinations**

Ischial Spines Large Average Small

Side Walls Parallel Convergent

Sacrum A. P. Curve Concave Straight Convex

Lat. Curve Concave Straight Convex

Notch Wide Average Narrow

Coccyx Movable Immovable

Straight Projects anteriorly

Diag. Conj.....cm. Not reached

Conclusions.....

.....*Examiner*

**Roentgen Measurements**

| INLET | MIDPLANE | OUTLET |
|---|---|---|
| A. P. .....cm. | A. P. .....cm. | Bituberal .....cm. |
| Trans.....cm. | Trans.....cm. | P. S.....cm. |
| P. S.....cm. | P. S.....cm. | |

**Pelvic Contours**

Inlet Symmetrical Asymmetrical

Ant. Segment Wide Average Narrow

Notch Wide Average Narrow

**Type of Pelvis** .....

**Comment** .....

.....

.....

.....*Examiner*

FIG. 9. Pelvic estimation chart.

*The Pubic Arch.* Palpation of the pubic arch with the thumbs is an important procedure. By this means the course of the pubic rami may be estimated. These bones form a typical arch in the female

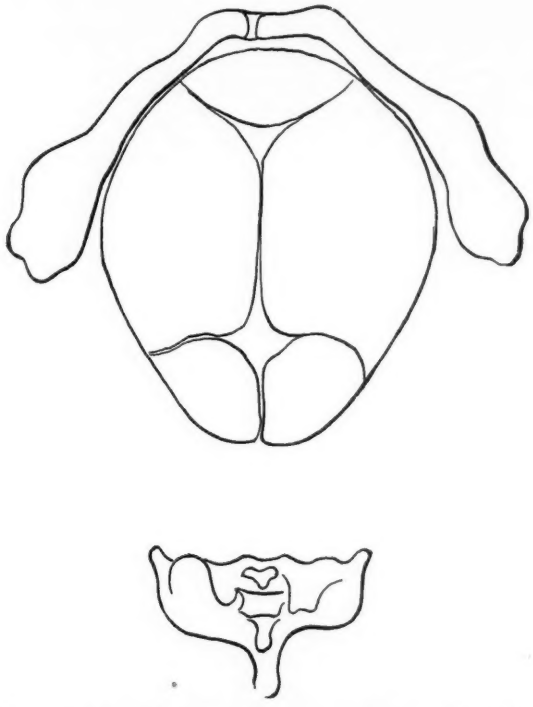


FIG. 10. Showing female type pelvic arch with fetal head fitting under symphysis. (From Thoms' "The Obstetric Pelvis," Williams & Wilkins.)

type, or their course may be straight, forming an inverted V in the male type. In the latter instance, the tubera ischii are usually not so widely separated and a funnel type of pelvic outlet may exist. In this latter type of pelvis it is obvious that in the process of birth the fetal head cannot fit up under the symphysis, and perineal lacerations and delayed labor may be frequently encountered. (Figs. 10 and 11.)

The width of the pubic arch may be roughly determined by palpatory methods. Some observers use the closed fist as palpatory mechanism to ascertain whether the arch is wide, average, or narrow. The bituberal diameter of the outlet may be measured with satisfaction by using the outlet pelvimeter, which was devised by the author some years ago. (Figs. 12 and 13.) The important thing

is to determine just where the end points of such measurement should rest on the tubera ischii. I find that there is no consensus of opinion in this matter. My own practice is to attempt to determine in each

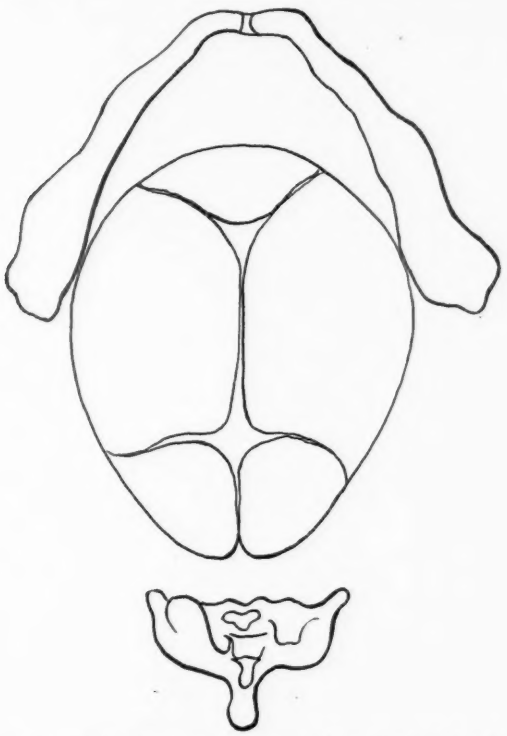


FIG. 11. Showing male type pelvic arch with fetal head forced posteriorly toward sacrum. (From Thoms' "The Obstetric Pelvis," Williams & Wilkins.)

case at which point on the inner edge of the backward curve of the tubera the fetal head might touch if impingement took place. As a rule these points are located at the lowest portion of the inner edge of the tubera ischii, assuming the pelvis to be resting with its tubera in contact with an horizontal surface such as obtains in the upright sitting position. The posterior sagittal diameter of this plane has been previously described and may be measured as shown, with the Thoms pelvimeter. The important thing in this latter procedure is to have the patient's hips well over the edge of the table so that the lower posterior surface of the sacrum may be easily palpated. As this diameter may be also measured roentgenologically, the two readings should check within reasonable limits.

*Ischial Spines and Side Walls.* These eminences are easily palpable on both rectal and vaginal examination. The interspinous diameter also may be roughly estimated, but the chief object of

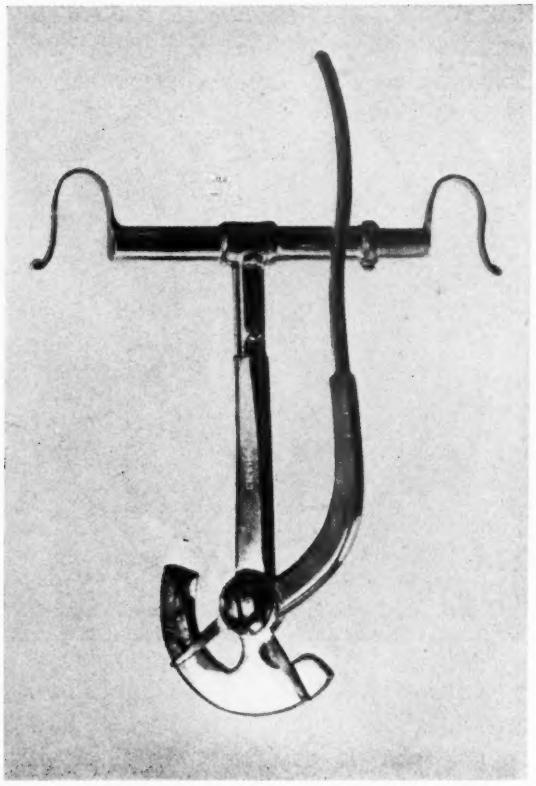


FIG. 12. The author's outlet pelvimeter. Half size. (From Thoms' "The Obstetric Pelvis," Williams & Wilkins.)

the internal examination is to determine the size and prominence of the spines in terms of large, average, and small. The male type of ischial spine conformation is almost universally large and prominent. The lower side walls of the pelvis are readily palpated, especially where there is narrowing or convergence such as is seen in male pelves and in certain pelves of the dolichopellic type.

*The Sacrum.* Careful rectal and vaginal palpation will determine a good deal of information concerning the anteroposterior and lateral curves of the sacrum. The former may be investigated by sweeping the examining finger upwards and downwards over the anterior surface of the sacrum and the lateral curve determined by a side to side motion over this surface. In this way we may find

out whether these surfaces are concave, straight, or convex, which may be important information in the diagnosis of abnormalities of the sacrum due to rachitic influence.

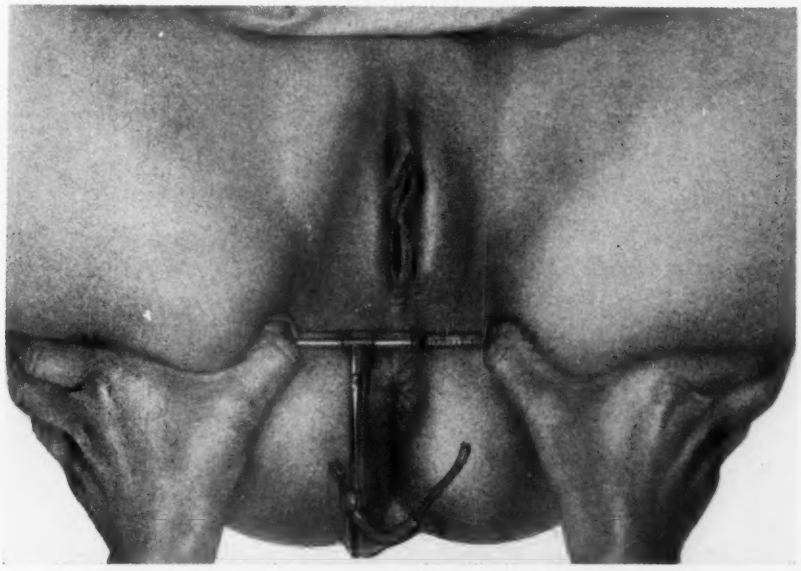
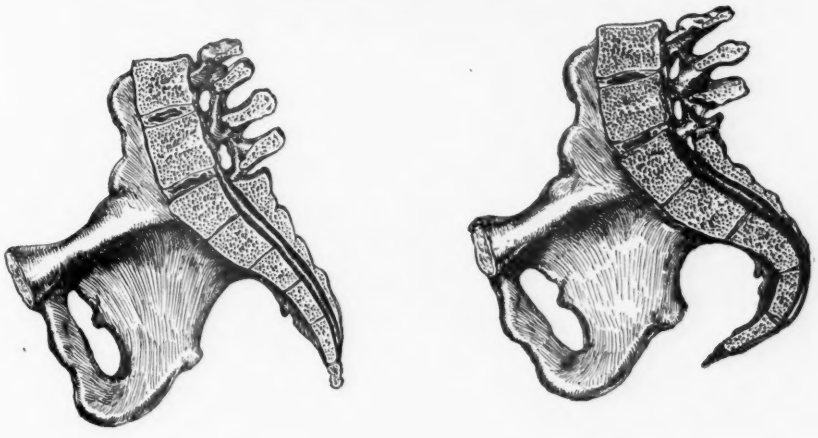


FIG. 13. Measuring of transverse diameter of outlet. (From Thoms' "The Obstetric Pelvis," Williams & Wilkins.)



A B

FIG. 14. Two types of abnormal sacral curvature. A, sacrum straightened out. May be entirely convex from above downward. B, abnormal concavity of sacrum. (From Thoms' "The Obstetric Pelvis," Williams & Wilkins.)

*The Sacrosciatic Notch.* This excavation may be palpated both vaginally or rectally. The latter procedure is probably the more useful. The strong ischiosacral ligaments may be readily felt and a

good idea of the character of the lower part of the notch be elicited, especially when abnormal narrowing is present.

*The Coccyx.* This bone is readily felt on vaginal and rectal

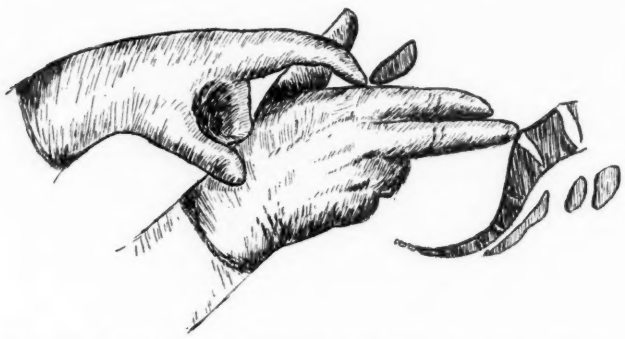


FIG. 15. Measuring the diagonal conjugate diameter. (From Thoms' "The Obstetric Pelvis," Williams & Wilkins.)

examination, and its relative movability established. If its position is a continuation of the downward sweeping curve of the anterior sacral surface it is said to be straight. If, however, the coccyx projects anteriorly, forming an angle with the tip of the sacrum, this is noted. The possibility of delayed labor due to forward malposition of the coccyx should not be overlooked.

*The Diagonal Conjugate Diameter.* The directions for obtaining this diameter are essentially as follows (Fig. 15): With the patient in the lithotomy position and prepared for vaginal examination, the examiner raises one knee by placing the foot upon a small stool. Then, sinking the elbow and resting it against the knee, the vaginal examination is made, using two fingers. The middle fingertip is pressed in the direction of the sacral promontory by means of steady pressure against the perineum. As soon as the middle fingertip touches the promontory, the internal hand is raised against the lower border of the symphysis. At this point the index finger of the outside hand rests on this latter point and the internal hand is withdrawn. The distance from the middle fingertip to the point marked by the index finger of the other hand is measured by calipers or tape to obtain the length of the diagonal conjugate diameter. DeLee states "After guessing at the inclination of the pubis and allowing for height, one deducts 1½ or 2 cm. or more, and thus approximates the length of the C.V. (true conjugate). Numerous fallacies underlie this reading, the compression of the fingertip, the bending of the joints, the slipping of the finger from the promontory, errors in

estimating the height, thickness, and angle of the pubis, etc." (Fig. 16.) To these I would add that in certain individuals, particularly primigravidae, the examination may be difficult because of

FIG. 16. Showing the effect of forward displacement and elongation of symphysis on the length of the diagonal conjugate diameter. (From Thoms' "The Obstetric Pelvis," Williams & Wilkins.)

the pain experienced by the patient due to the rigidity of the perineal soft parts. More important than any of the above fallacies, is the fact that in many pelves the sacral promontory is so high above the posterior point of convergence of the iliopectineal lines that the reading becomes practically valueless for obstetrical calculation. In these cases, as has been pointed out previously, the plane of the inlet or true obstetrical inlet is not encroached upon, or influenced by, the promontory of the sacrum; hence any anteroposterior diameter using the promontory as an endpoint may be misleading.

After these objections, one may wonder why any attempt at all is made to measure this diameter. The author's feeling is that while making the routine vaginal examination to elicit other information, reasonable attempts should be made to reach the promontory also. If it is accessible, this measurement may be determined, but if it is

not reached readily, pronounced attempts to do so should not be carried out and the designation "not reached" should be entered on the outline chart. So many factors, however, result in an inability

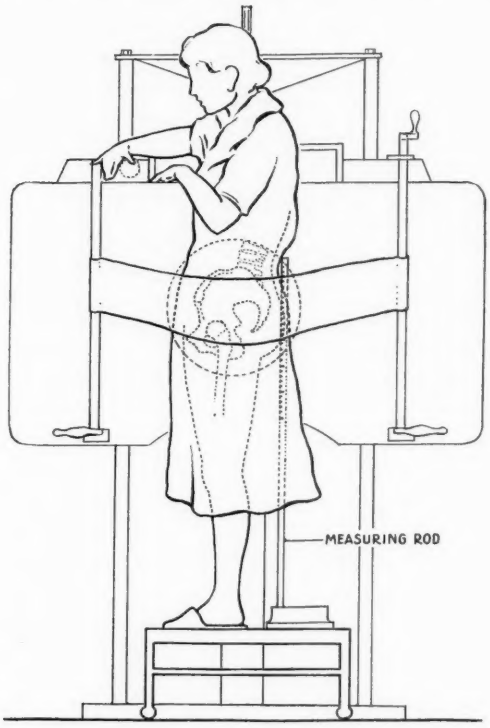



FIG. 17A. Position of the patient for lateral pelvimetry. (From Thoms and Wilson, in *Yale J. Biol. & Med.*, 10: 437, 1938.)

to reach the sacral promontory that by no means should the designation "not reached" be construed as indicating that the anteroposterior diameter of the pelvic inlet is adequate for obstetrical purposes.

*Conclusions.* Under this heading the examiner briefly notes any abnormalities or variations from the normal, also his opinion of the adequacy of the pelvis in its entirety.

### ROENTGEN METHODS FOR PELVIMETRY

The author's first communication on the subject of pelvimetry appeared in 1915, and in 1922 the results of his first experiments with Roentgen pelvimetry appeared. From then until the present, the results of his experience in applying Roentgen methods to clinical problems have appeared from time to time. In 1938, in collaboration

with H. M. Wilson, certain Roentgen methods were described which are useful for routine obstetrical pelvimetry, and these techniques with slight modifications may be described again here.

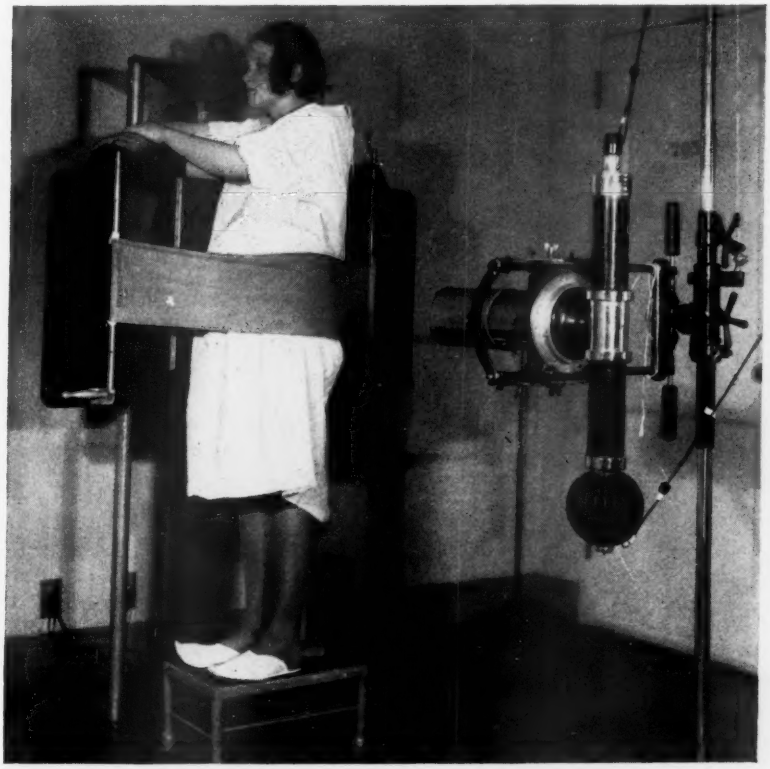


FIG. 17B. Position of the patient for lateral pelvimetry. (From Thoms and Wilson, in *Yale J. Biol. & Med.*, 10: 437, 1938.)

For routine purposes we take two views: one of the pelvic inlet by the so-called grid method, and one lateral projection. It has become obvious to us that an adequate survey of the pelvis for obstetrical purposes is not possible without the employment of both techniques. Furthermore, inasmuch as the mensuration of the antero-posterior diameter of the pelvic inlet may be determined in both views, each procedure becomes an excellent check on the accuracy of the other. Our routine is to take the lateral projection first, and this technique may be described as follows:

1. The patient removes her clothing, putting on heelless slippers and the usual hospital bed-gown open at the back.

2. She is placed standing in front of an erect Bucky diaphragm or an adjustable cassette changer, such as is used for chest work, with

either the right or left lateral aspect of the body toward the target. (Fig. 17.) The arms are folded across the chest.

3. The target film distance is 40 inches and the target is centered

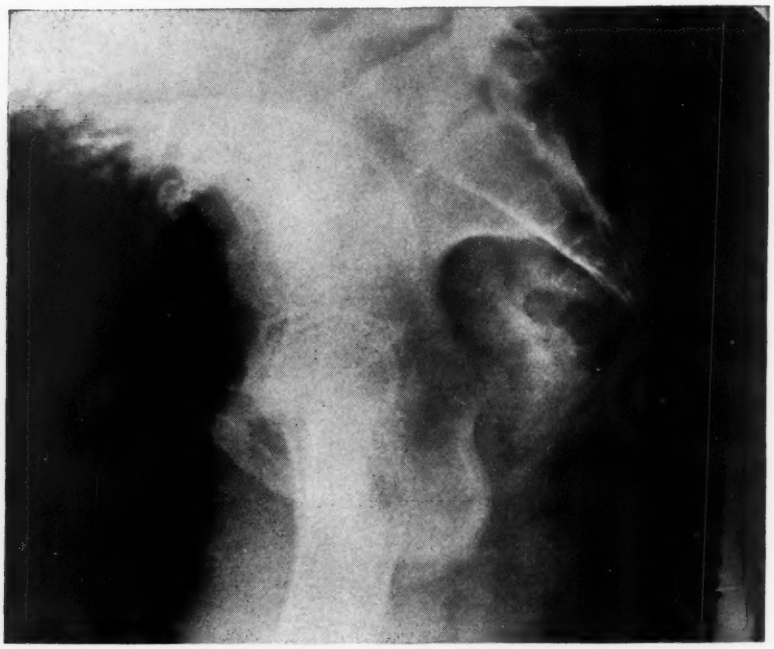


FIG. 18. Lateral roentgenogram at term. Fetal head shown above the pelvic inlet. Evidence of slight disproportion is present. Corrected centimeter scale at side of film, by means of which all anteroposterior diameters may be measured. (From Thoms and Wilson, in *Yale J. Biol. & Med.*, 10: 437, 1938.)

at a point on the external conjugate diameter one-third the distance from the symphysis pubis to the depression under the fifth lumbar vertebra. A 60 inch target film distance may be used, employing a Lysholm grid instead of a Bucky diaphragm, as shown in Figure 17. The shorter distance with the Bucky diaphragm seems to give somewhat better definitions in our hands.

4. A binder is placed around the patient and attached to the cassette changer to insure further steadiness during the exposure.

5. Before the exposure is made, an upright metal rod (lead and iron) with a centimeter scale perforated in a lead strip is placed posterior to the patient close to the fold of the nates. This rod is somewhat similar to that described by Weitzner for use in lateral horizontal position.

6. The time of exposure varies with the thickness of the patient, all other factors being constant; in general the time is from twelve to fifteen seconds.

*Comment on Lateral Technique.* When developed and viewed, the following landmarks should be readily identified: anterior and posterior borders of the symphysis pubis, acetabula, ischial spines, ischial tuberosities, the lower lumbar vertebrae, the promontory and anterior surface of the sacrum, and the sacrosciatic notch. (Fig. 18.) On one edge of the film may be seen the shadows cast by the perforations giving corrected centimeters in the sagittal plane of the patient. By means of calipers, using this scale, any diameters in this plane may be measured.

CHART OF TECHNIQUE FOR LATERAL PELVIMETRY

| | |
|---|---|
| Kvp | 80 |
| Ma | 30 |
| Dist | 40 inches |
| Bucky diaphragm | Yes |
| Time | Chart |
| Screens | HiSpeed |
| Medium sized cone | |

THICKNESS TIME-CHART

| Centimeters | Seconds | Centimeters | Seconds |
|---|---|---|---|
| 27 | 9½ | 33 | 15 |
| 28 | 10 | 34 | 17 |
| 29 | 11 | 35 | 19 |
| 30 | 11½ | 36 | 21 |
| 31 | 12 | 37 | 23 |
| 32 | 13 | 38 | 25 |

The advantages of this lateral technique may be summarized as follows:

1. All the anteroposterior diameters of the bony pelvis may be measured, including those of the pelvic inlet, midpelvis, and inferior strait. The anterior and pelvic inlet diameters of the important midpelvic plane are readily determined.

2. The contours of the anterior surface of the sacrum may be studied, a matter of importance in the recognition of sacral abnormalities, especially those due to the influence of rickets.

3. When lateral roentgenograms are made at term or in labor the relation of the presenting part to the superior strait may be studied with advantage. Since early in labor, in the majority of

instances, the fetal head approximates the L.O.T. or R.O.T. position, it will be seen that it is possible to measure also the all important biparietal diameter of the fetal head. In cases where the so-called trial of labor is being given and the patient is having frequent uterine contractions it may be more convenient to use the lateral horizontal position. When this is done we use the same distance, reduplicating as far as possible the relationships which we have used for the same patient in the erect lateral projection.

4. In certain instances where diagnosis is doubtful respecting the presence of multiple pregnancy or fetal abnormality, lateral roentgenograms may give more information than that obtained by the usual anteroposterior technique.

The advantages of this present modification appear to be as follows:

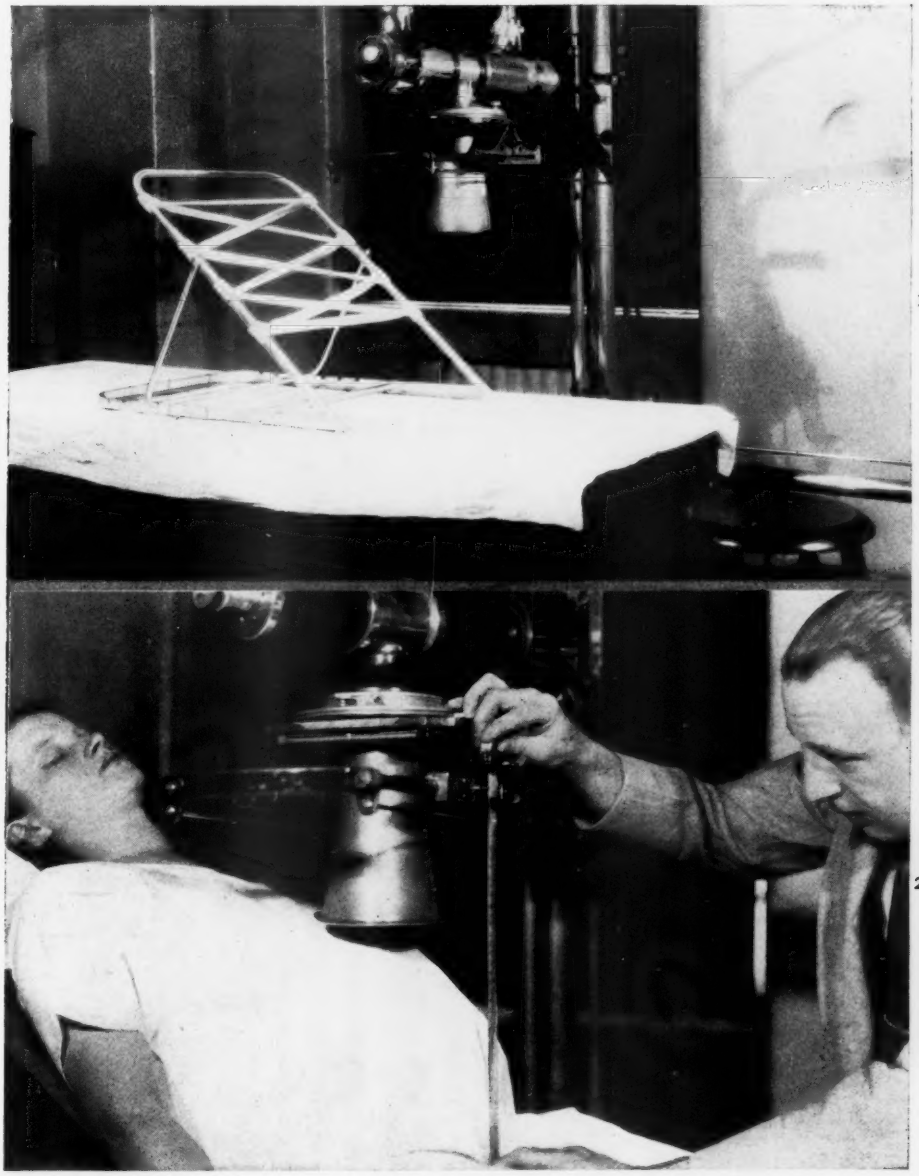
1. Decreased distortion. The use of a 40 or 60 inch target film distance for projection of the sagittal plane lessens the distortion due to divergence of the *x*-ray beam. This is especially true in large subjects whose lateral pelvic diameter results in a long object film distance. The erect posture also decreases distortion resulting from rotation of the pelvis on its vertical axis and tilt on its anteroposterior axis. With the subject horizontal on a flat table it is difficult to secure good superimposition of the shadows of the two innominate bones. The erect position not only corrects this, but permits a more satisfactory projection of the lumbosacral relationship. The sag of the lumbar spine, due to the disproportion between the pelvic and shoulder diameters when the patient lies on her side, is not met in the erect position.

2. Corrected centimeter scale. The projection of a centimeter scale corrected for the sagittal plane of the individual pelvis appears on the edge of the finished film. The obstetrical conjugate is determined by selecting a point on the cortical white line of the posterior wall of the symphysis 1 cm. below the superior border and measuring with calipers the distance between this point and the upper sacrum at the point where the iliopectineal lines meet.

3. Simple equipment. The erect posture in lateral pelvimetry makes possible a long target film distance. No special apparatus is required other than the perforated opaque scale and the Bucky diaphragm or Lysoholm grid.

The centimeter grid method for pelvic inlet pelvimetry may be described in its essentials as follows:

1. The patient is placed on the roentgenographic table in a semi-recumbent position which is maintained by a backrest shown in Figure 19. (The lower third of this rest is cut away in order that



19

20

FIG. 19. Back rest used for pelvic inlet pelvimetry.
FIG. 20. The superior border of the symphysis is located. (From Thoms and Wilson, in *Yale J. Biol. & Med.*, 10: 437, 1938.)

the lower part of the patient's back may be exposed.) In placing the patient in position we endeavor to make the pelvic inlet of the

pelvis as nearly horizontal as possible, with the patient in a comfortable, unstrained position.

CHART OF TECHNIQUE FOR SUPERIOR STRAIT PELVIMETRY

| | |
|---|---|
| Kvp | 80 |
| Ma | 30 |
| Dist | 30 |
| Bucky Diaph | Yes |
| Time | Chart |
| Screens | HiSpeed |
| Medium cone | |

THICKNESS TIME-CHART

| Centimeters | Seconds | Centimeters | Seconds |
|---|---|---|---|
| 24 | 1 | 32 | 8 |
| 25 | 2 | 33 | 9 |
| 26 | 3 | 34 | 12 |
| 27 | 3½ | 35 | 14 |
| 28 | 4 | 36 | 17 |
| 29 | 5 | 37 | 20 |
| 30 | 6 | 38 | 23 |
| 31 | 7 | | |

2. The level of the pelvic inlet above the sensitive film is established as follows: (a) By means of calipers the vertical distance is measured from some point on the table top to a point on the anterior surface of the symphysis pubis 1 cm. below its superior border, or this point in space may also be determined by measuring downward with a tape from some point on the tube-stand. (Fig. 20.) (b) By means of calipers the distance is determined from the interspinous space between the fourth and fifth lumbar vertebrae, as determined by palpation, and the table top. (Fig. 21.) For practical purposes, an imaginary line drawn between the posterior point on the body and the point on the upper and anterior surface of the symphysis will bisect the plane of the pelvic inlet.

3. The tube is centered in the midline about 6 cm. posterior to the upper border of the symphysis and the exposure is made.

4. The patient is removed from the table, the tube and exposed film remaining *in situ*.

5. The centimeter grid, a lead plate with perforations exactly 1 cm. apart, is introduced into the same plane as that previously occupied by the pelvic inlet (Fig. 22), as determined by the caliper readings and the measuring tape, and a second (flash) exposure made on the previously exposed film.

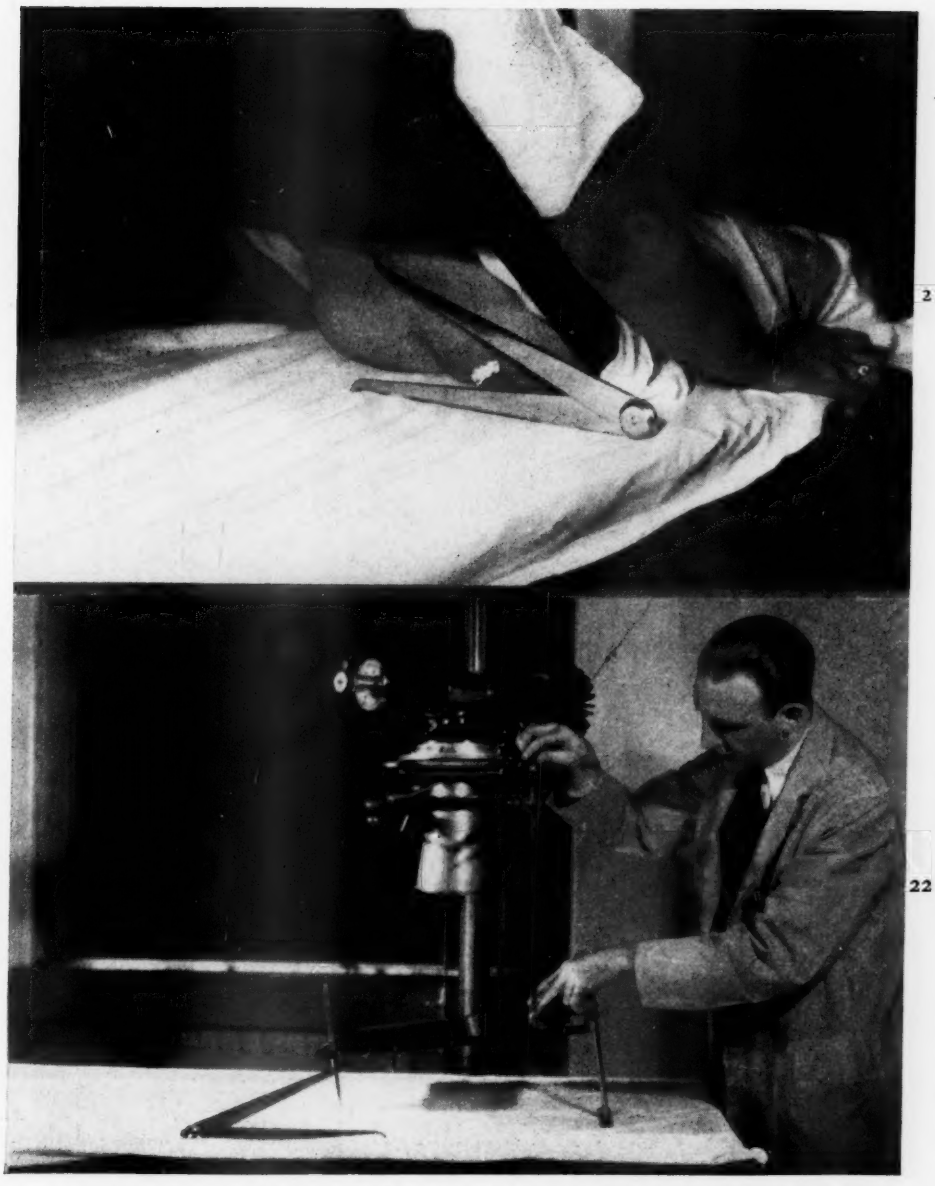


FIG. 21. The interspace between the fourth and fifth lumbar vertebrae is located.
FIG. 22. The centimeter grid is placed in the plane formerly occupied by plane of the pelvic inlet. (From Thoms and Wilson, in *Yale J. Biol. & Med.*, 10: 437, 1938.)

On viewing the developed film an outline of the pelvic inlet is shown, together with shadows produced by the perforations in the lead plate. (Fig. 23.) The distance between these projected dots

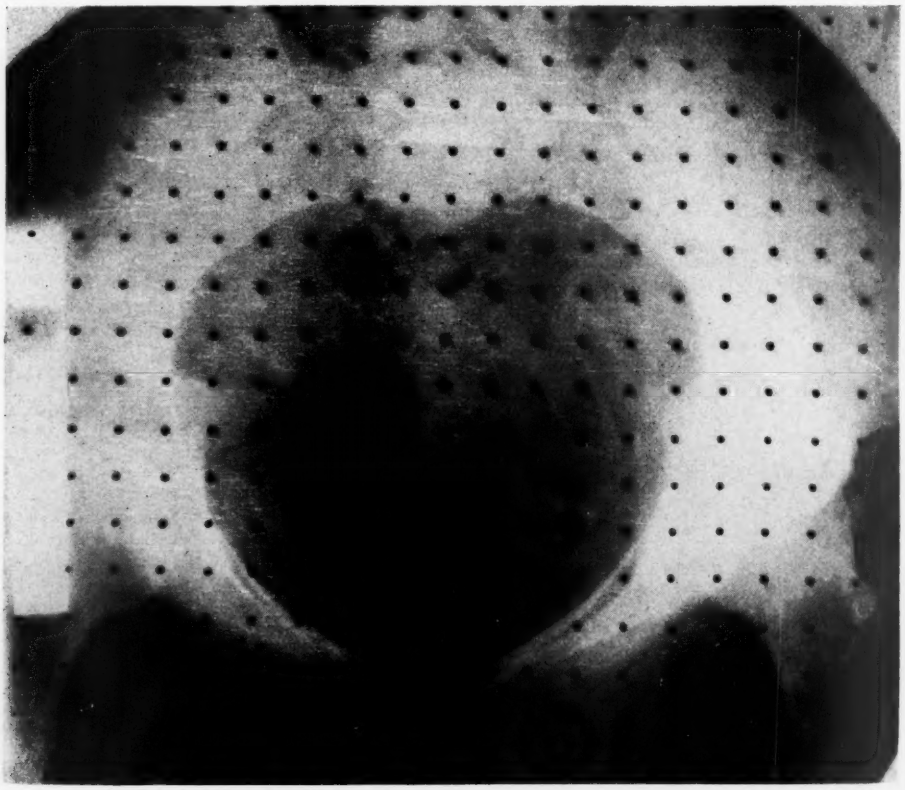


FIG. 23. Outline of pelvic inlet. Distance between dots represents centimeters in this plane. The anteroposterior diameter should check with that of the lateral projection. The bispinous diameter also may be measured in this film by correcting for the plane in which these processes rest (see text). (From Thoms and Wilson, in *Yale J. Biol. & Med.*, 10: 437, 1938.)

represents centimeters in the plane of the pelvic inlet. Not only may the anteroposterior and transverse diameters of this plane be read directly, but a reduced outline of the pelvic inlet may be drawn on centimeter paper and this record filed with the prenatal record. The latter procedure is but a matter of reduction and the simple apparatus which we use for this purpose has been recently described.

*Comment on Centimeter Grid Technique.* For obstetrical and investigative purposes we have no reason to doubt the accuracy of our results. On numerous occasions we have performed experiments,

both with dried pelves and in the living woman, in which our results have been checked at laparotomy. Interesting in this latter connection is the recent communication of Dr. E. A. Schumann, who, in

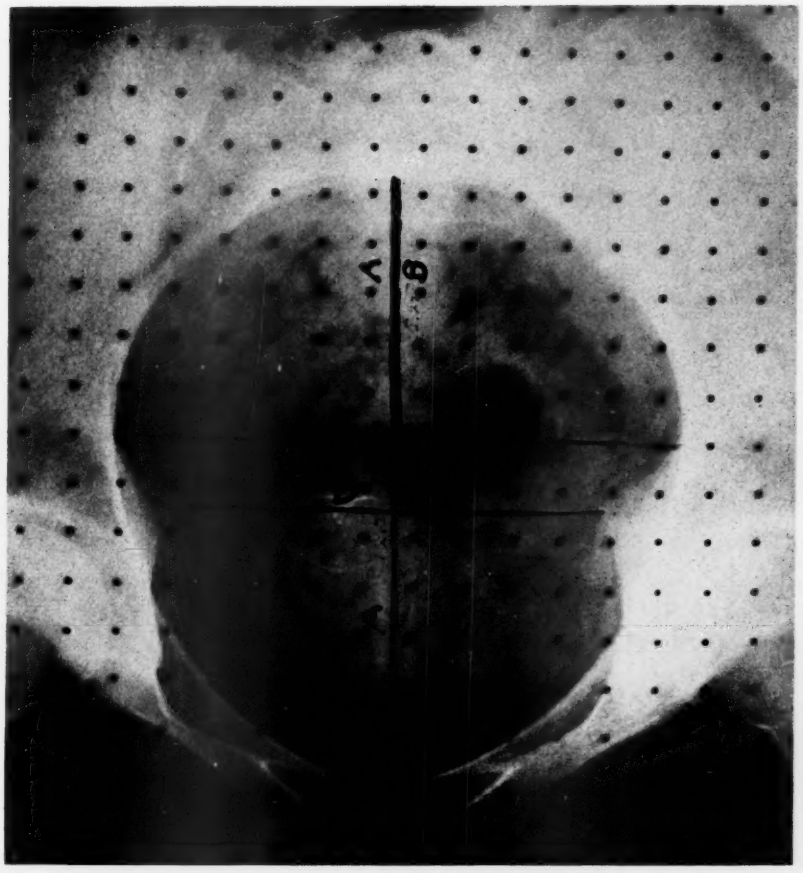



FIG. 24. Film of pelvic inlet. Lines indicate cardinal diameters: A, anteroposterior diameter of inlet. B, posterior sagittal diameter of inlet. C, transverse diameter of inlet. D, transverse diameter of midplane.

1936, measured fifty women directly at laparotomy and observed: "In order to determine the accuracy of roentgenologic measurement, a group of the foregoing patients were subjected to measurement by the technique of Thoms, and it was extremely gratifying to learn that the diameters of the inlet as determined by direct intra-abdominal measurement corresponded in every instance with those obtained by this technique, the variation being constantly less than 2 mm."

The question naturally arises as to whether the posterior point on the patient's body (i.e., the interspinous space between the fourth

and fifth lumbar vertebrae) is too indeterminate for the placing of the grid in the plane of the pelvic inlet. Our experiments with dried pelves have shown us that this posterior point may vary as

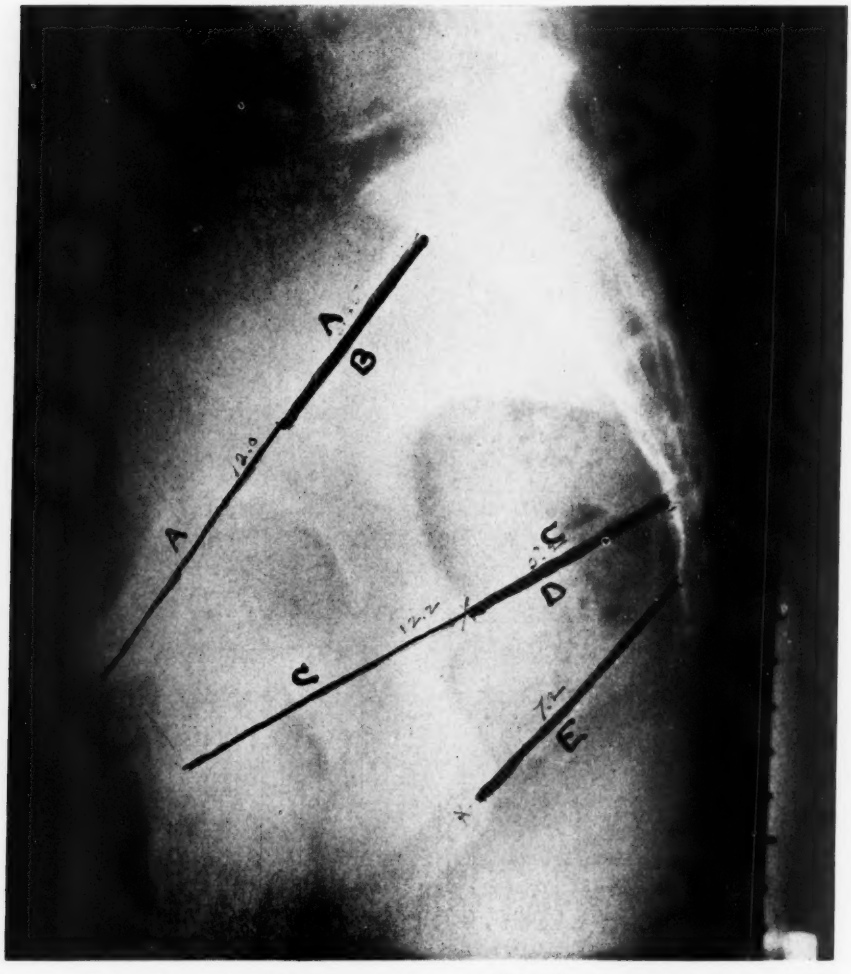



FIG. 25. Film of lateral pelvis. Lines indicate cardinal diameters. A, anteroposterior diameter of inlet. B, posterior sagittal diameter of inlet. C, anteroposterior diameter of midplane. D, posterior sagittal diameter of midplane. E, posterior sagittal diameter of outlet.

much as 2.5 cm. above or below the ideal situation, without measurably affecting the mensuration as recorded on the film. In other words, there is a leeway of at least 5 cm. in determining this point. In any event, the lateral projection should leave no doubt as to the true anteroposterior diameter of the pelvic inlet and the centimeter grid projection should check this within 2 mm.

Furthermore, it is entirely possible, by developing and viewing the lateral projection before placing the grid in position for exposure, to determine exactly its ideal position. This occasionally may be necessary for exact results in rare instances of marked sacral deformity where a double promontory or other malformation places the obstetrical conjugate in a plane much different from that of the anteroposterior diameter of the superior strait. When it is desired to regulate the placing of the grid in this manner, this may be accomplished by projecting a line on the lateral film from the posterior border of the symphysis through the plane to be measured. With the calipers (using the corrected scale) we measure downward at a right angle to this line to the tip of the spinous process of the last lumbar vertebra as seen in the projection. With the patient in the sitting position we measure the distance of this spinous process above the table top and the data are complete for the exact placing of the grid. However, for routine purposes we are confident that this refinement in technique is not necessary, for when the lateral projection is used as a check, any discrepancy of the anteroposterior diameter in the centimeter grid projection will be manifest.

*Summary.* Among other features which impress us in using these methods may be mentioned the fact that the roentgenograms so obtained are readily interpreted by both the obstetrician and the roentgenologist. The simplicity of these methods, using but two 10 × 12 films and consuming a minimum of time for the technique, reduces the expense of Roentgen pelvimetry to a very reasonable sum. A further reduction in overhead expense is possible by grouping patients to be done at one time. The use of the lateral technique before and during the so-called "trial of labor" is of great advantage in studying the cephalopelvic relationship.

It is important to mention again the mensuration of the diameters of the midpelvis and pelvic outlet, since these portions of the pelvis play a very definite part in dystocia. The distance between the ischial spines may be measured in these films by first noting the distance of these processes below the anteroposterior diameter on the lateral film and then measuring the interspinous distance on the inlet film, correcting by means of a table showing the spread of the rays at the level of the plane in which these processes rest.

## CONCLUSION

A little over two years ago the author published a paper dealing with the uses and limitations of Roentgen pelvimetry, and much

that was said at that time bears repetition here. The experience which has come from the use of newer methods of measuring the pelvis has taught us that we are in a transitory period from the general use of older and somewhat approximate methods to the adoption of newer and more accurate procedures. These newer methods have not only furnished new concepts as to the female bony pelvis but also more knowledge of cephalopelvic adaptation during the process of birth.

In the present monograph, which concerns itself chiefly with the estimation of pelvic capacity, it would be helpful indeed to consider in further detail certain clinical relationships which pelvic capacity may affect. At the present time, however, it may be said that many such important relationships are not completely established and may become so only through further clinical experience. The reason for such discrepancies is apparent when we consider the many factors that are concerned in the process of birth. I have previously commented upon this matter as follows. "The birth canal is composed not only of the bony structures but of soft parts, and the fetus also is composed in the same way. Mensuration of these bony structures therefore is not an entirely complete survey of the birth passage and its passenger. The forces which are concerned in labor are subject to influences which render them both voluntary and involuntary, and it would appear that much of this influence is hormonal in character. In addition, the influence of such factors as race, constitution, disease, emotion, and pain all share in the complex process. The usefulness of the knowledge of the dimensions of bony structures of the pelvis and fetus, therefore, has certain limitations, and to draw definite conclusions as to the outcome of labor without considering the whole picture is not only hazardous but unscientific. In other words, the greatly useful knowledge which these Roentgen methods has given us must be properly assimilated with our greatest therapeutic weapon, namely, clinical experience. It is not enough to visualize the pelvis and fetus in space by means of roentgenograms. Indeed it is easy for the inexperienced and enthusiastic to be sidetracked into false security if too much emphasis is given to Roentgen information so obtained. Roentgen methods are of great value, but they must be properly interpreted."

Again it is the author's wish to emphasize the importance of the routine use of the measures recommended in primigravid women. An unsuspected midplane or an outlet contraction may be present in the primigravid patient at term with adequate inlet capacity in whom

the fetal head is engaged, and there is no way of accurately determining such occurrence without Roentgen means. Furthermore, the occurrence of wide variations of pelvic form in presumably normally formed individuals is definitely a matter of concern to the obstetrician, and only by Roentgen investigation may these variations be established. With regard to the use of these methods in the presence of pelvic contraction, comment seems hardly necessary. Dippel in a recent comment upon this question has ably stated the correct scientific viewpoint. "It may be argued," he writes, "that there are so many factors in labor, namely, the size of the baby and the character of the uterine contractions, that an approximation is enough. We do not agree with this viewpoint. The very fact that these other factors are difficult to evaluate accurately makes it all the more important, in cases of contracted pelvis, that we should have as precise information as possible concerning the one factor that we can measure with precision."

In conclusion, the author wishes to emphasize that at the present time we possess methods for measuring the female pelvis which are quite adequate for obstetrical purposes and by their routine use we are bound to enrich our knowledge and experience for the betterment of scientific obstetrics.

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